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ANXIETY STATES*

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CLINICAL conditions which are essentially anxiety states, and conditions in which anxiety, while not the primary factor, plays a significant part, constitute an outstandingly large part of medical practice. Our knowledge of the anxiety states has been considerably enlarged in recent years by the introduction of a number of new concepts, certain of which will be discussed.

Autonomous states.—It is now recognized that a certain number of the anxiety states may become self-perpetuating (Cameron, 1944). Once an individual has been rendered anxious over a prolonged period, he tends to become over-reactive to situations likely to produce anxiety. Hence, even when the major primary stress which was responsible for his original anxiety has been removed, the minor stresses and tension-producing situations of day-to-day living, by repeatedly eliciting the anxiety response, are sufficient to maintain his over-reactivity and, therefore, his anxiety state. A concrete illustration is afforded by the individual who has been exposed to excessive and prolonged anxiety, either in combat or in the form of financial or job insecurity.

The recognition of these states has been delayed largely because, in medicine, we tend to take over the rather simple nineteenth century physics concept of causation: Cause A produces Effect B; when you stop A, you stop B. Or, still more literally, you put the poker in the fire and it gets hot; you take it out and it cools down to its original temperature.

When this simple, straight line idea of causality is applied to the anxiety state, it expresses itself like this: put sufficiently severe stress upon an individual and he will become anxious; remove the stress and his anxiety will disappear. But the fact is that in a proportion of cases this does not happen, the patient continuing to remain anxious and fearful about many things long after the original stress has gone. From this there has arisen a series of unfortunate consequences. The first is that his relatives, neighbours, and too often his physician, have said: "Everything is all right now. You have nothing to complain about", thereby simply increasing the burden which he has to carry. A second consequence is their assertion that since everything is all right—since the dangerous war service is over, since his financial worries are at an end—the real cause of his complaints must be something that earlier medical examinations had overlooked: an impacted wisdom tooth, a histamine sensitivity, his sinuses, his stomach, bowels or hæmorrhoid tag. There then ensues that unhappy pilgrimage through the offices of the internist and the surgeon, the orthopædist and the urologist.

There are some aspects of the behaviour of living organisms which follow the system of straight line causality, but for the most part behavioral trends follow other more complex types of causality, of which autonomy is one. Here event A produces event B, which produces C, the latter producing D and D finally producing A. In other words, you have a circular rather than a straight line system of causality.

Our methods of breaking up such autonomous systems are still quite elementary. They are based either upon the principle of damping down the reaction, by chemical sedatives such as the barbiturates or physiological sedatives such as insulin, in the anticipation that if it does not occur over a period, the tendency will disappear, or upon the principle of breaking up the system, the only example of this being the drastic procedures of lobotomy.

Closely related to this concept of the autonmous anxiety state is that of the subliminal tensional rise. There is growing appreciation of the fact that the level of tension fluctuates

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in the individual from minute to minute, depending upon the demands which the environment make upon him. By far the larger number of such fluctuations are subliminal—we do not become conscious of them. But in the anxiety neurotic, whose tensional level is persistently high, even such small increments may suffice to produce symptoms. Hence the probable explanation of the sudden appearance of symptoms which because of their apparent causelessness are specially disturbing to the patient, who, being unable to discover what evokes them, cannot take steps to prevent them.

In actuality, one may presume that he has been reacting to an incident, such as walking in a crowded street, meeting a stranger, sitting in a movie or occupying a seat in a bus facing a number of other passengers. All such incidents tend to cause subliminal tensional rises in the normal individual; in the anxiety neurotic, such rise may constitute a sufficient increment to evoke his anxiety pattern.

Differentiation of anxiety.—This is a second concept of major significance in the development of our knowledge of anxiety. It is generally agreed that the anxiety response is total, *i.e.*, that the whole organism responds. It is not so clearly recognized that the manifestations of tension and anxiety tend to vary in their intensity from one part of the organism to another, and that the pattern which this variation takes, while tending to remain constant for the individual, differs considerably from person to person (Cameron, 1944). Failure to recognize this differentiation of patterns continues to lead to a most considerable number of faulty diagnoses. The explanation of by far the greater number of these errors is the same, namely, that the diagnostician has concentrated his attention upon the patient's complaint, to the exclusion of the patient. Hence we still find an extraordinary number of hypertensive, gastric ulcer or headache patients treated with little or no attention to any other part of the patient lying outside the area of complaint. In passing, one may comment that this trend increases with specialization. The oldtime family doctor was too versed in human nature to fall into some of our modern errors of exclusion.

Certain further comments should be made upon this concept of the differentiation of anxiety. As a working tool, we now rely on the premise that if an individual is repeatedly exposed to long and severe anxiety, that area in which the anxiety expresses itself predominantly may become progressively disorganized with each repeated period of anxiety. Hence we see that the man who from earliest childhood has tended to react to stress by anorexia, flatulence and perhaps vomiting, tends to pass over gradually to ulcer formation and eventually to reach a point where medical and surgical measures have to be brought in to supplement psychiatric therapy.

We are inclined to think that the same is true of certain cases of arterial hypertension and perhaps of hyperthyroidism. In regard to the latter, it may be stated that a certain percentage of anxious people tend to show increased activity of the thyroid gland during stress, and that when the stress is removed, the elevated basal metabolic rate and other signs of hyperthyroidism disappear, to return, often with increased intensity, with subsequent exposure to stress, until a chronic hyperthyroidism results. If, instead of removing the stress, we remove the thyroid itself, the results can only be designated as most unfortunate.

When we ask the obvious question why one man reacts to stress by losing his appetite and another by developing feelings of pressure around his chest, we are entering a region where there are more unexplored than known areas. In a certain number of cases, the pattern is due to the significance which it has for the individual. The child brought up in the home in which excessive attention is paid to eating—"You must eat the right amount at the right time", or conversely, "If you don't do this, you won't be allowed to eat your supper"—is apt to use his eating function as a means of asserting himself, and from this it is only a step to the establishment of gastro-intestinal symptoms as a reaction to stress. Similarly, the child brought up in a home in which the father suffers from a cardiac condition will soon attach excessive significance to precordial twinges and pressures.

There remains, however, a large number of cases which cannot be explained upon this basis. Current attempts to relate the differentiation of anxiety patterns to personality characteristics—traits of repressed hostility being related to gastric ulcer—seem without adequate foundation.

We have referred to the fact that the pattern of tension and anxiety varies from individual to individual. Reference must also be made to the fact that though these patterns are relatively fixed, some degree of variation within a given person does occur. One of the major factors in bringing this about is the immediate need of the individual. Jacobson (1928) in a series of valuable investigations has shown that when an individual undertakes a task in which he has acquired skill, tension tends to fall in those parts of the organism which are not involved, remaining at a high level in those parts which he requires for the performance of his work. A simple illustration is afforded by the typist in whom tension remains high in the arms, shoulders and neck but falls in the lower extremities. Observations which are of direct clinical significance are that when the individual is pushed to perform at a greater speed than is customary, this differentiation gradually breaks down and there ensues a general tensional rise, which increases to the point where he is pushed completely beyond his capacity and becomes so disorganized that he gives up. In this connection it may be stated that the tensional anxiety neurotic shows a considerably reduced capacity to differentiate tension during work, and hence his level is likely to remain uniformly high.

Residual tension.—This is a concept which has been well known to the experimental psychiatrist and psychologist for at least two decades, but is one which we have hardly begun to apply clinically. Tension can be observed and measured in a variety of ways—the blood pressure, heart rate, gastric activity, and changes in the electrical potentials of skeletal musculature. The latter is one of the most convenient. It has been shown by Sharp (1941) that when work has been undertaken, the tension in the muscles involved does not subside immediately, or in an even manner. After a period of two minutes' work with a dynanometer, the tension in the arm muscles of his subjects did not return to resting level for about thirty minutes, and during that time of resting showed notable increases and decreases. Clinically, we are aware that the capacity to relax varies considerably from one individual to another. We are also aware that many modern industrial occupations are extremely tension-producing, and we are increasingly inclined to suspect that those individuals who show difficulty in getting rid of tension should not be allowed to enter certain types of occupation, as for instance the highspeed, repetitious jobs.

When this concept of residual tension is linked to that of the differentiation of tension, certain possibilities of great interest present themselves. Clinically, we have seen a number of patients in whom certain areas appear to remain chronically over-reactive and which serve, as it were, as "trigger" zones which, once stimulated, set off a generalized tensional anxiety response. We have conjectured that either the tensional level remains high in such cases, or that there are areas, specially reactive to stress, which rapidly develop high tensional levels, which then spread to produce a general tensional anxiety state.

In some individuals, these areas exist in the skeletal musculature or in parts of it. We have learned to distinguish two large areas which appear to be particularly over-reactive. The first is located in the head and neck and consists in a special liability to develop aching of the neck, head pressure complaints, difficulty in swallowing, constriction of the throat, circumoral tremor, voice tremor and stuttering. The second is located in the lower extremities and shows itself in the form of increased deep reflexes, clonus, jerking of the legs in going to sleep, Rombergism, and feelings that the surroundings are swaying. In other individuals, we suspect that areas of the smooth musculature, such as the urinary bladder, may constitute trigger areas.

Experimentally, we have been able to show (Cameron, 1947) that once an area, such as the cardiovascular system, has been rendered over-reactive by means of large doses of adrenalin, it remains so for several days, and if stimulated again during that period not only shows itself to be much more reactive than before but also serves to elicit a generalized tensional anxiety state.

Thus far, the working premises which have been stated are those which seem designed greatly to expand our knowledge of and our control over these widespread and crippling states of anxiety. At this point, I should like to urge in the strongest possible terms the destruction and removal from our thinking of a concept which has a great share of the responsibility for our failure adequately to recognize, and hence to deal competently with,

these states. This is the concept of the imaginary complaint.

As you know, if a patient complains about a certain area, and if careful examination of that area reveals no deviation from the normal, it is customary, sooner or later, depending upon the conscientiousness of the physician's examination, to dismiss the complaint as imaginary. It is difficult strictly to define what he means by imaginary, perhaps because at this point he has given up being a scientist and is now a moralist. Among other things he means that the patient should not be complaining and that he could help it if he wanted to.

Now this serves simply to indicate our deep confusion concerning psychological states; for if there is a complaint, that is as definite a phenomenon as a mass in the breast or a bulging saphenous vein. As we know, it can be at least as crippling, and it most certainly deserves a scientific explanation.

The reasons why a patient may complain about an area in which no deviation can be found are numerous. He may complain because the area has a special significance for him, as in the case of a woman who had a great desire to talk about certain matters concerning which she felt guilty and an equally great fear that she might get into trouble if she did so. Her complaint was of tightness of the jaws. Or a patient may complain because he feels anxious and socially insecure and needs to know that he is the object of regard. Or again, he may complain because of the need for expressing deep hostility.

And finally we may say that all tensional anxiety states are total responses, and hence there is a great range of happenings throughout the body which may result in symptoms appearing, the physiological correlates of which we have not yet recognized. One may point to the fact that the aching in the back of the neck, so common in these conditions and so often dismissed as imaginary, almost always is accompanied by increased tonus in the musculature of that area, a fact which can be recorded by means of the electromyogram to the satisfaction of the most concrete-minded of men.

One may also point to the fact that a general hyperæsthesia is a common accompaniment of tensional anxiety states, and that we can point to a lowering of the sensory threshold as an explanation of certain of the more unusual complaints of throbbing in the abdomen, photophobia and complaints concerning visual disturbances and skin sensations.

Throughout this discussion of tensional anxiety states there is implicit the concept that anxiety is the response of the individual to stress and primarily to environmental stress. But since, as indicated earlier, the organism reacts as a whole, peripheral participation in the form of muscular tension or over-reactivity of other organs tends to facilitate central responsiveness, hence setting up a reciprocally reinforcing system. If this can be interrupted at any point, the action breaks down. Hence the value of such diverse agents as psychotherapy, chemical and physiological sedations, and lobotomy.

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RÉSUMÉ

L'importance de l'angoisse, dans les états où elle est un facteur primaire, mais aussi là où elle vient s'ajouter, en les compliquant, à d'autres états, tient à ce qu'elle tend à se perpétuer. Au contraire des phénomènes de la physique, elle ne disparaît pas avec une cause simple; l'état anxieux reconnaît plusieurs s'influençant réciproquement et formant cercle. Les moyens d'attaque de ce cercle vont de l'usage des sédatifs à la lobotomie pré-frontale, la psychothérapie occupant une place intermédiaire mais non la moins importante. Les manifestations cliniques de l'angoisse se différencient à l'extrême d'un malade à l'autre; elles sont la pierre d'achoppement de ceux qui entendent traiter le symptôme en oubliant de soigner le patient. Dans ces état de choses, celui-ci subit des thérapeutiques dont ses maux ne sont pas justiciables, et le répit accordé l'angoisse causale donne libre cours à la désorganisation de plus en plus complète du secteur, somatique ou psychique, dans lequel l'angoisse s'était manifestée d'abord. Ce désordre principal s'ajoute à un substratum de tension généralisée dite "résiduelle", qui se traduit dans les musculatures striée et lisse, et rend le malade sensible à des incitations qui seraient sub-liminales pour le sujet normal. L'hypertonus des masses lombaires, chez l'anxieux qui a mal au dos, atteste bien de la réalité concrète d'un symptôme qui, pour être purement fonctionnel, ne mérite pas d'être appelé imaginaire.

PAUL DE BELLEFEUILLE

CONCRETE BLOCKS SUGGESTED IN PLACE OF METAL HOSPITAL BEDS.—Concrete blocks in place of metal beds in hospitals of the future are recommended by a French architect. Jean Walter, a French hospital designer, explains his novel concrete bed in a report on hospital building in the British journal, *The Lancet*. Patients would sleep on the usual mattress and springs. Instead of the metal frame support, the bed would be a hollow concrete block covered with earthenware. "This would save considerable trouble in cleaning," M. Walter points out. Each block would have drawers for the patient's property and medical equipment, and the concrete bed could be wired for diagnostic instruments.

THE SCIENTIFIC OUTLOOK*

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THOSE of us who can look back upon fifty years in our profession have been privileged to witness greater advances and greater changes in outlook than has occurred in any other half century.

When first I joined the profession as a medical student in 1897, surgery was entering upon its boom period. Prior to 1847 surgery must have been a grim procedure. Used only as a last resort, it called for great manual dexterity and self-confidence to complete an operation in the minimum of time, while to operate upon a shrinking and probably a shrieking patient, required an iron nerve not rendered too susceptible by unnecessary sympathy or imagination. Even when anæsthesia had removed the pain of the actual operation, there remained the grim prospect of hospital fever, until Lister's great discoveries in the 1860's made it possible to minimize, if not completely to remove this.

Lister's work was, I think, the first great medical advance founded upon the discoveries of a worker in another branch of science. Other men before Lister were as near as he to a clinical understanding of the problem, but he was fortunate in following Pasteur, who gave him the key which he employed so skilfully to elucidate not only this surgical problem, but to establish a basis for the study of many other conditions. When I entered the medical school in 1897, Lister's antiseptic methods were accepted the world over. A new race of surgeons had been bred into the profession with other requisites than mere speed and self-confidence. The work of these men was founded upon a science, bacteriology, and although this was a very small branch in those days it turned the surgeons' minds into scientific channels.

Bacteriology, upon which antiseptic surgery was based, was at this time in its infancy, and was included in the teaching of general pathology. I think I am right in saying that at that time we recognized only one streptococcus and two staphylococci and about a dozen other organisms. I remember well the pride with which our professor of pathology exhibited to us the first specimen of the plague bacillus, which he had obtained from a sailor at the new Manchester dock. Now bacteriology is a science in itself, with numerous branches. Not only do we know of large numbers of organisms undreamed-of when I was a student, but other distant relatives, the viruses, etc., while general principles of antibodies, immunity, etc., have gradually been worked out.

It was about this time that Roentgen discovered his magic rays which made it possible for the surgeon to see the bones whose positions, up to now, he could recognize only by palpation. The first x-ray apparatus in my teaching hospital was established only in my late student days and when I was a house surgeon the x-ray examination of fractures was by no means routine, and we were taught still to rely upon our clinical senses. It was not long before the clinician and the scientist combined to discover opaque substances which made the study of the alimentary canal possible, while at a much later period substances were discovered which would be excreted to make visible changes in kidneys, gall bladders, etc., or could be injected into Fallopian tubes and internal spaces.

In my student days the rising generation of surgeons had grasped the inter-relation of science with surgery, and the necessity for the young surgeon to be well trained in the sciences, and for the older ones to keep abreast of scientific advances. Without a sound basic knowledge of chemistry, physics, biology, bacteriology, pathology and above all of physiology, no surgeon could hope to take a part in the thrilling exposure of nature's secrets, on which the surgeon and his allies were at that time engaged. I remember seeing the first cystoscope used in Manchester, though I am afraid none of us, not even the surgeon, at that first attempt saw anything but a bright red field: this again was the combined effort of the surgeon and the scientist.

Surgery, at that time, was beginning to blossom. Month by month, almost day by day, new organs and new pathological conditions were found amenable to the knife, but only those surgeons who were abreast of the developments in physiology and the basic sciences could plan advances in surgery. In other words, a surgeon

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now had a scientific outlook, not merely that of a craftsman.

In the early years of this century, when I was a hospital resident, aseptic replaced antiseptic surgery. This new development came from the United States, and reached us in the first decade of this century. In 1903-04 when I was a house surgeon at the Manchester Royal Infirmary, we used sterile gowns, the first autoclave sterilizer having been installed when I was a student, but we had no gloves nor masks nor caps, and we still used strong antiseptic lotions for our instruments and hands and for the patients' skin. A year later, when I had transferred to Saint Mary's Hospital for Women, asepsis came in with a rush, and about the same time came the Trendelenburg position, which simplified access to the pelvic organs. Asepsis was a great advance, as organs could now be handled more delicately and without irritation, and it was this which made possible the future great extension of surgery. In those days there was much discussion about sepsis and its avoidance, and even more on the treatment of cases we had infected. Today it is taken for granted that a clean case will remain clean.

When we speak of the limitation of the old-time surgeons however, we must bear in mind the development of theatre equipment which today we take for granted. Metal tables capable of many movements; special lighting; autoclaves for dressings and instruments; dressing rooms for surgeons, most of these the combined invention of the surgeon and the scientist.

When first I went to Saint Mary's Hospital, the last few weeks of the old hospital, the operating theatre was a small room with a wash hand basin in one corner, a gas ring in another over which the Sister boiled her instruments, a solid wooden table incapable of any movement, and no artificial light. I remember on one occasion a sudden thunderstorm blotted out the light in the middle of a vaginal hysterectomy, but the surgeon, unperturbed, continued by the light of a bicycle lamp, a reading lamp and a candle lamp which three students held.

Since that time surgery has developed out of all recognition, and is now divided into as many special branches as before there were operations, and calling to its aid many discoveries undreampt of 50 years ago, transfusions, chemo-therapeutic agents, radium, x-rays and many other wonderful inventions all based upon science.

In the early days of this century, when surgical development was in full tide, almost all ambitious students wished to become surgeons or specialists in a branch in which surgery was used, gynæcology, ear, nose and throat, ophthalmology etc.; a number of able men, repelled by the long and irregular hours of the clinician or attracted to pure scientific research, entered one of the many scientific branches, physiology, pathology, bacteriology, biochemistry, etc., though in my early days many of the chairs in anatomy, physiology and pathology were filled by disappointed men; anatomy by surgeons, physiology and pathology by physicians who had been overlooked in the fierce competition for clinical appointments in teaching hospitals. Before the sudden development of surgery through its partnership with science, medicine had attracted the best brains, and medical knowledge, in its clinical aspect, developed in the 18th and early 19th century more rapidly than did surgery. In the early part of this century however, pure medicine was rather in the doldrums. As students we disliked the idea of spending an hour to examine a patient and to make a diagnosis, only to be told that the only treatment was Mist. Arsenicalis or some placebo, whereas, so it seemed to us, the surgeon would, in the same time, have removed the offending organ. Later in the century medicine was to make a handsome recovery when it too wedded science. Take only two conditions which, in the early part of the century were always fatal; diabetes, the cure of which was discovered in this city, and pernicious anæmia, now cured by liver extracts. I am not sure, but I always think that medicine recovered its position after the first world war, which was fought for over four years in the worst possible conditions without any serious outbreak of typhoid fever, whereas in the South African War, only fifteen years earlier, our men had died like flies from this scourge.

These developments in medicine, like those in surgery, have come about by the co-operation of the clinician and the scientist, and nowadays the physician must have a scientific training and a scientific outlook.

In my own branch, obstetrics and gynæcology, the story runs parallel with that of surgery, as they both base their modern de-

velopment upon Listerism. As so much of British obstetrics was domiciliary it took a much longer period for antiseptic principles to permeate the whole practice, but it gradually did so and this, with the recognition of oral infection and the more recent discovery of chemo-therapeutic agents is the main factor in the reduction of maternal mortality from 5 per thousand as it was at the end of the first Great War, to 1.2 per thousand as it stands today.

In Great Britain gynæcology was developed chiefly by the obstetrician, although a few pioneers are best described as general surgeons with a leaning to gynæcology. These obstetricians adopted the antiseptic and later the aseptic technique as did the general surgeon and it was really the gynæcologist who showed that the abdomen could be opened with safety. In the last fifty years the changes in gynæcological practice have been enormous, as the gynæcologist, like the general surgeon made use of the discoveries of the scientist.

Probably the greatest field of scientific research in my department has been the discovery of the hormones. When I was a student we knew much about the thyroid and a little of the suprarenals, and we knew vaguely that sex characteristics depended upon the sex glands. Now we are flooded with information about these organs and the various hormones produced and the symptoms which are produced by too much or too little of some particular extract.

Clinically we are still groping in the dark. It may satisfy the scientist to know that the increase or decrease of a certain substance will produce certain symptoms. Unfortunately, these same symptoms may be due to the increase or decrease of certain other substances, and we clinicians are sometimes puzzled to know whether in certain cases to give more of one hormone or less of another. We may be sure, however, that before long this tangle will be straightened out by the combined effort of clinician and scientist.

I think all will agree that the clinician and the scientist combined have developed our profession in a way impossible to either alone, and that the medical student must be given a sound foundation in the basic sciences while the practitioner, whether he be physician, surgeon, obstetrician, specialist or general practitioner, must maintain a scientific outlook.

While maintaining a scientific outlook we must, however, be careful not to forget our main calling, to cure the sick and to give aid to the suffering. We must not allow our scientific outlook entirely to eliminate the humanitarian or to teach our students that patients are so many rabbits for experimental purposes or labelled with certain diseases calling for stereotyped treatment. Each patient is an individual reacting differently, in minor degrees, to the same disorder, and in a large degree to the same treatment and that each is a study in himself.

In England we had before this second Great War very few whole time chairs in clinical subjects. During the war an interdepartmental committee known as the Goodenough Committee was set up to study medical education. On this subject of whole time chairs they heard much evidence for and against, and wisely reported that the system should be tried in a few universities. In Great Britain by whole time chairs we mean that the professor devotes his whole time to what you call ward patients and is debarred from attending private or semi-private patients. I imagine the individual members of the Committee had in mind well trained and experienced clinicians with a scientific outlook and with problems to elucidate which the strain of private practice prevented them from working upon. There are a number of these men, but few of them can afford in the full tide of private practice, and with the commitments they have undertaken, to abandon these for the salaries which the universities offer. A salary adequate for a man who has deliberately chosen in youth the more leisurely and more certain path of academic teaching and research, cannot attract the man who is in the full tide of success after the long hard years of fierce competition which the academic teacher does not experience. There are a few instances where first-class experienced clinicians have made this sacrifice, but they can only be few, the field with the present small salaries being generally restricted to the young, partly trained and completely untried, or older men who have not made a success of clinical work.

The universities, however, are in full cry after the words "research" and "academic outlook". To many of them research begins and ends in a laboratory, while academic outlook often means little more than a spirit of contemplation which requires double the time to do any piece of clinical work. They overlook the fact that clinical observations are as

much research as are those in the laboratory, and that the best is one who correlates the two. Laboratory technique can be acquired in a comparatively short time and competent technicians command comparatively low salaries; the well trained clinician is the product of many years of careful training, and is under a strain which University men do not understand. If the clinician has had laboratory training, as so many clinicians now have, it is an asset, but all that is essential is sufficient scientific knowledge to direct his laboratory technicians and to correlate their findings with his clinical observations.

More dangerous, because more subtle, is the encouragement given in Great Britain to men to train for University posts through whole time appointments without contact with patients in private practice, and without the stimulus of competition with their rivals. However desirable it may be for an older man to have leisure to digest the accumulated facts of a busy clinical period and to correlate them to scientific research, a young man with limited hours of duty, unhurried time for contemplation and clinical experience limited to one social class in an institution, may develop little more than a habit of contemplation.

The Universities overlook the fact that in England our pioneer work was done by clinicians with or without laboratory facilities, and generally by clinicians working in the full tide of practice, whose time for research had to be snatched in small sections or by means of rigid organization, and which because of this restriction must be used to the greatest effect.

Our knowledge of the nervous system has made enormous advances. Much of this is due to the laboratory work of the physiologists, but much more to the clinical physician and most to the neuro-surgeon, such as Harvey Cushing and his successors, who in this generation have built up a new branch of surgery which has brought great blessings to mankind as well as additions to our scientific knowledge. Another branch of modern development is orthopædics, developed in England after the first World War, around and upon the work of that great clinician, Robert Jones.

Lister was a clinician and would not have grasped the adaptability of Pasteur's discoveries to surgery if he had been a pure scientist. Mackenzie, who transformed our knowledge of heart disease, was a clinician and a hard working general practitioner during the time he made his greatest contributions to our knowledge.

The ideal professor of a clinical subject is a trained clinician with a scientific mind, one who can work with the scientist and understand his findings even if he is not able to carry on his own laboratory experiments. What seems to be futile is to appoint to these posts men with poor clinical training, as this cannot be supplied by clinical assistants without loss of prestige to the professor and moreover he, the head of a clinical department, is unlikely to appoint assistants with a better clinical training than himself and, if by accident he does so, it is unlikely that he would accept their guidance.

It should not be overlooked that one of the main functions of a professor of a clinical subject is to train students and that, unless he is a well trained clinician himself, his department will be a failure. It is right and proper to bring up the student with a scientific outlook, but he must be trained first and foremost to treat patients and it is wrong to give him the idea that scientific methods of examination can ever be more than aids to his clinical observations and examination.

What makes matters worse with us is that the English universities cannot offer salaries which compare with the income a good man would make in clinical practice, due in large measure to the understandable objection of distinguished professors in other faculties to professors of clinical medical subjects receiving so much more than they themselves do.

The reasonable answer would seem to be to allow the professor of a clinical subject a limited amount of private practice, which would not only increase his income, but keep him in touch with all classes of the community. This might entice good, well-trained men old enough to have proved their worth. As it is, young, untried men poorly trained in clinical medicine or older men who have failed as clinicians may be the only applicants, whose academic outlook may manifest itself in an inability to work more than from 10 a.m. to 5 p.m. and the ability to spend one or two hours in making a diagnosis which a trained clinician would do in half an hour.

What the answer will be I do not know. Some of the whole time men complain of the salaries they receive, and some threaten that they will throw up their posts and go into practice.

Another danger we must guard against in our teaching is to rely too much upon scientific aids. By all means have x-ray and all laboratory examinations done, but let these be adjuncts to clinical observation and examination. It is only by careful observation that the great clinician is made. We have all suffered from the enthusiastic radiologist who makes the diagnosis; what the clinician wants is a meticulous record of what the x-ray shows, he will make the diagnosis by fitting the x-ray and other tests into his own observations.

What is even more important is the danger that the scientific outlook may obscure the human approach: that we may be interested in our patients as scientific curiosities rather than as human beings. I think I can best illustrate what I mean by what I have observed in obstetrics. From 1905 to 1908, for three and a half years, I was resident obstetric surgeon to our large and ancient women's hospital in Manchester, Saint Mary's Hospital. In this post apart from patients in the hospital I was responsible for about 4,000 confinements in the patients' own homes. These confinements were attended by a staff of midwives who lived each in her own small district, but I took the responsibility for these patients during the confinement and for a month before and a month afterwards. Midwives are a class neither you nor any other Dominion nor the United States have much experience of.

At that time midwifery was scandalously poorly paid, and these women, therefore, were drawn from a social stratum little above that of their patients. They were poorly educated, most of them were married and had families of their own to look after. Their training in midwifery consisted of attendance at a course of lectures at our own hospital, attendance at a number of confinements with another midwife, and the passing of an examination held by this hospital.

In the theory of their profession and in antisepsis they left much to be desired. But in clinical knowledge they acquired great skill, and during the whole period for which I was responsible it was seldom that any of them overlooked an abnormality or failed to notify me. Springing from a class little higher than their patients, they understood their domestic difficulties, and did not expect preparations beyond the patients' capability to provide. They devoted themselves body and soul to their patients, answering the first call, remaining with them throughout the whole labour, or returning at frequent intervals. Having families of their own they understood the natural trepidations of a primigravida facing the great unknown, and gave her confidence and encouragement when she required it. They knew all the little tricks for relieving discomfort and pain, and altogether were veritable sheet anchors to which their patients clung.

In 1902 the first Midwives Act was passed, though it did not come into full action until 1905. Under this a Central Midwives Board was set up to take responsibility for the training and examination of would-be midwives and for the supervision of midwives in practice. By easy stages the training and examination was increased, as was the pay and social position, and gradually the pupils were drawn from higher social classes with a much higher standard of general knowledge. Today the midwives are a highly trained, well educated body of women, who have received a long training and passed stiff examinations, and their salaries are very different to my midwives of forty years ago.

But with it something is missing. Instead of a middle aged woman with a family of her own, the pupil is young, unmarried, and most often a generally trained nurse who takes a scientific interest in her work. Her early training is entirely in hospital under Sisters, highly trained but unmarried and with such a scientific outlook that they are interested chiefly in the abnormal, or in the normal only when birth is imminent. In the first stage, the time when there is much discomfort, fear and anxiety, the patient is often left alone or in the company of one or two patients in the same state. A young probationer looks in from time to time to see that nothing seriously abnormal is happening, but she does not know how to give that confidence and encouragement so much needed at that time, while many Sisters only take a serious interest when this stage is over and the patient removed to the labour room for the actual birth. From a statistical standpoint these patients do well, but often there is a psychological scar. There is in-

creased scientific interest but a little more humane interest would not be amiss.

Forty years ago the training of medical students in obstetrics was lamentable. In the subsequent period this training has been greatly improved, as it has been with you but during this same period more and more of the normal midwifery practised in Great Britain has fallen into the hands of the midwives. You do not employ midwives, a doctor is engaged to attend each confinement, and I presume is present throughout the first stage, the time when his personality will be of the greatest benefit to his patient.

One other drawback to institutional obstetrics is the elimination of the husband. I believe that the anxiety through which the husband passes when he is in the house and frequently sees his wife, especially in the first stage, brings a realization of what the birth has meant to his wife, in a way impossible with delivery in an institution which excludes the husband. Moreover, having been about during the whole process he feels that the child is more a part of himself than when it is handed to him fully launched upon the world some ten days or a fortnight later when his wife leaves the hospital. If the anxiety of labour is to remain the domestic cement which I believe it was intended to be, I believe we must either retain domiciliary midwifery or arrange for the husband to be admitted more freely to the institutions.

I have taken the midwife as an example not as the only type whose training has overstressed the scientific outlook. The same criticism may be made with the training of nurses in our general hospitals; much theory, a scientific outlook, sometimes forgetfulness that the patient is an individual, helpless and in distress, to whom the handing of a bedpan when called for may give more relief than the most scientifically carried out dressing.

I wonder if I dare quote an example told to me only a few weeks ago. A well known Englishman sent to the U.S.A. on a mission last year was found on the day he was to return to have a high temperature, and to be suffering from pneumonia. He was treated with the greatest care and consideration and transferred to a most expensive room in one of the best known hospitals. He was kept waiting some little time while the room was prepared; then, put to bed in comfortable surroundings, he snuggled down with the one desire gratified, to lie undisturbed and at rest. But no, hardly had he got comfortably settled than two nurses appeared and forced him out of bed because they had forgotten to take his weight during the waiting period. No doubt a highly important scientific detail but one he would gladly have dispensed with.

Let me take one other and last example. One of the greatest advances in gynæcology in the last half century has been the improved treatment of cancer. When I was a resident a woman with cancer of the cervix was doomed. Then came Wertheim's hysterectomy, by which we were able to obtain about 40% alive at the end of five years of those in an operable stage. Some years later came radium, giving similar results but without the same postoperative discomfort, and applicable to more advanced cases. A tremendous advance in forty years, but no thoughtful man can be satisfied, or would dare to say that any individual case will be cured by either treatment.

We know, and the laity know, that a patient suffering from cancer is liable to recurrence no matter how early the disease may seem, and no matter what the treatment. I believe very strongly, therefore, that a patient should not be told the diagnosis, unless there is some definite business reason, and that he or she should be treated in the general wards mixed with other patients, and not segregated into special hospitals or wards where he will be surrounded only by patients suffering from this disease and some in advanced stages. Under these circumstances the patient who eventually proves to be cured is constantly looking for symptoms of recurrence, and life becomes a constant anxiety. During my active career I made it a rule to treat all my cancer cases, by operation or by radium, in the general wards mixed with other patients, and I was especially careful to treat here, with radium, some patients admitted for functional hæmorrhage, as it was a proof that radium was not used only for cancer.

Just before the war our Government passed a cancer act. The war has prevented its application, but soon it will be in action. A great drive will be made to educate the people about the early signs of cancer, to persuade

them to come for diagnosis to cancer clinics, and to believe that early cases are curable.

The bureaucrat appears to class cancer with tuberculosis, of which early cases can usually be cured. Cancer is different: the earlier the case the more likely is the cure, but even under the most favourable circumstances this is far from certain. Some day a cure or preventive will be found. Until then we shall produce untold misery by encouraging cancerphobia and by segregating cases of this condition for treatment which we know in our heart of hearts will cure only a certain percentage. Here again the scientific outlook is overlooking the humane.

SUMMARY

In the last half century very great advances have been made, and what is of greater importance, we have developed a scientific outlook. It is most important that this continues and that it is used as a basis of the instruction of medical students. It has, however, its dangers, and we must be careful in our practice, and especially in our teaching and training of students, nurses and midwives, that this scientific does not entirely submerge the human outlook.

Science has done much, and will do more, but the main object of the clinician, his students, and above all his nurses and midwives, is the treatment of human beings, to cure them of their ills, to remove their fears and anxieties, and to relieve them of their pain and distress.

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PROBLEMS OF THE ANÆSTHETIST—HAZARDS IN THE USE OF ANÆSTHETICS*

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ONE of the factors in the development of anæsthesia as a distinct specialty in recent years was the realization of the countless hazards that a patient is subjected to in receiving any anæsthetic. Fifty years ago, the administration of ether was believed to be practically without risk. Since then tremendous progress has been made in the demonstration of hazards inherent in the administration of the various anæsthetic agents, and in the development of newer agents that are potentially less harmful to the patient.

Elam published statistics which show that in Great Britain the death rate from anæsthesia has actually increased as additional agents and techniques have been introduced. Part of the explanation for this anomalous situation lies in the fact that many untrained physicians have attempted to utilize these more modern techniques such as intravenous anæsthesia, cyclopropane with carbon-dioxide absorption, or high spinal anæsthesia, with disastrous results. They simply were not aware of the hazards of these techniques, which the trained anæsthetist would know how to avoid or treat should they occur.

All anæsthetics, regardless of the mode of administration, are potentially hazardous. The hazards involved in the use of any anæsthetic agent are influenced by such factors as, (1) nutritive and metabolic state of the patient, (2) technique or mode of administration, (3) dosage of drug, (4) duration of anæsthesia, (5) nature and site of operative procedure, and (6) the skill and knowledge of the anæsthetist.

General anæsthesia.—The changes produced by the general anæsthetic agents in the central nervous system are of a reversible, although as yet unexplained nature. As long as adequate oxygenation of the tissues is maintained, we are not damaging the nervous system to any detectable degree. Unfortunately, the same generalization cannot be made when the other systems of the body are being considered. The avoidance of hypoxia then may be regarded as the cardinal principle to be observed in the administration of any anæsthetic. Failure to do this can produce changes in all organs of the body. The central nervous system, particularly the cerebral cortex, is most readily damaged by impairment of its oxygen supply. Despite some clinical evidence to the contrary, it would appear most hazardous to restrict the intake of oxygen below 20 to 21%. Healthy robust individuals may tolerate an oxygen intake of as low as 14%, but the uncertainty of the tolerance makes this an exceedingly dangerous state to induce intentionally in any patient.

More recently the importance of the efficient elimination by the body of carbon dioxide

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during general anæsthesia has been brought to our attention. Retention of carbon dioxide in the tissues may result from such factors as the use of partially exhausted soda lime in the absorption technique or from depression of respirations, the tidal volume being inadequate to ventilate properly the lungs with consequent gradual build-up of carbon dioxide in the body. Any dead space in the apparatus permits carbon dioxide accumulation, as does any degree of rebreathing in the absence of soda lime. The presence of a high oxygen tension in the tissues and blood interferes with the elimination of carbon dioxide from the tissues.

The toxic effect of increased carbon dioxide may manifest themselves in a variety of ways. The earliest evidence is an increased rate and depth of respirations, increased pulse rate, and elevation of the blood pressure. Waters believes that convulsions under general anæsthesia are frequently associated with carbon dioxide retention. Under cyclopropane, carbon dioxide "build-up" may occur due to the depressed respirations and high oxygen percentage in the inhaled mixture. When the anæsthetic is discontinued, allowing the rapid blowing-off of the carbon dioxide, there results a marked fall in blood pressure. This has been termed "cyclopropane shock" by Dripps.

In the presence of such potent respiratory depressants as cyclopropane and pentothal sodium, the respiratory signs may be completely masked, allowing alveolar concentrations of carbon dioxide to exceed 10 to 12%. Beyond this concentration, carbon dioxide is a depressant to the respiratory centre. These toxic manifestations of carbon dioxide serve to impress upon one the importance of maintaining an adequate tidal volume during all anæsthesias, and the strict avoidance of excess dead space in the apparatus.

Changes in blood.—The changes that occur in the formed elements of the blood constitute no danger to the well-being of the patient. The increased white blood count is a transient phenomenon occurring to some extent with all inhalation agents. The changes in chemical constitution of the blood by some general anæsthetic agents may be a source of grave danger, particularly to the handicapped patient—the diabetic, or dehydrated patient in a state of acidosis or impending acidosis. Hyperglycæmia occurs in normal persons under ether anæsthesia; in diabetics there is an even greater elevation. Insulin is believed to be no longer effective in lowering the blood sugar in patients under ether. The mobilization of glucose results in a marked decrease in the heart muscle, striated muscle, and liver. It is agreed that ether is contra-indicated in the diabetic patient. Hence in the presence of diabetes, it is advisable to use agents that are less apt to produce marked hyperglycæmia, such as cyclopropane and nitrous oxide mixtures.

The tendency to produce acidosis by ether constitutes a further hazard to diabetic and dehydrated patients. Stehle and Bourne have demonstrated the discharge of phosphoric acid from the muscles under ether anæsthesia. This results in a diminution of the alkali reserve with a tendency to lowering of the blood pH.

The production of acidosis under cyclopropane is due to a somewhat different mechanism. Seevers *et al.* have shown that the carbon dioxide retention that occurred with cyclopropane due to a diminished respiratory exchange, resulted in a shift of the blood reaction towards the acid side. With the maintenance of adequate tidal volume and reduction in the dead space preventing the accumulation of carbon dioxide, this tendency to acidosis may be prevented to a considerable degree. Hence this need not constitute a dangerous factor in the anæsthetizing of the dehydrated, acidotic patient.

Liver.—The liver plays a two-fold rôle in the evaluation of any anæsthetic. The agent may either act directly on the liver in a deleterious manner or the liver may serve as a detoxifying base. Chloroform produces the greatest liver damage of any anæsthetic agent. Rosenthal and Bourne have demonstrated in dogs that chloroform always produces liver damage, and certainly this seems to apply equally to humans. Chloroform produces acute fatty degeneration with a rapid depletion of the liver glycogen.

Ether produces only transient mild impairment of liver function. Other inhalation agents have likewise been shown to produce little or no change if administered in the absence of hypoxia. Ravdin and co-workers were unable to demonstrate definite clinical evidence of liver damage by divinyl ether. Nevertheless they advised against its use in the

presence of known biliary tract disease. Bourne and Raginsky found no impairment of function of the normal liver following the administration of avertin fluid. The use of smaller doses of avertin was recommended in the presence of impaired liver function of moderate degree, to avoid prolongation of the period of narcosis since the liver is the site of detoxification of tribromethanol. The use of pentothal sodium in the presence of hepatic dysfunction does not appear to constitute any appreciable danger. Masson and Beland concluded from their investigations that pentothal sodium is detoxified in all tissues of the body, and not to any great extent in the liver. Nevertheless caution should be exercised in the administration of either of these agents in the presence of a diseased liver.

Kidneys.—Kidney function undergoes marked depression during anæsthesia regardless of the agent. This effect would appear to be due to extra-renal factors, rather than to any intrinsic damage to the kidneys by the anæsthetic used. Barbour and Bourne believed that the oliguria found with ether was due to the hæmoconcentration. Studies by Coller and Moyer and co-workers with cyclopropane and ether have led to the conclusion that the depression of function is due to such factors as changes in acid-base balance, ionic distribution, and altered cardio-circulatory function. Coller, discussing this experimental work, stressed the dangers in the administration of normal saline during the anæsthetic and in the immediate postoperative period, in the absence of gross loss of electrolytes through vomiting or gastro-intestinal drainage. In the presence of depressed kidney function, the administration of excess saline adds a further strain on the kidneys leading to retention of the saline with production of tissue œdema, impaired wound healing, etc. Glucose solutions are given to promote diuresis.

Cardio-circulatory system. — To remain in common usage, an anæsthetic agent must have relatively no toxic effects on the heart. Transient alterations in the rhythm may be tolerated providing that it can be shown that they do not have a fatal termination nor show evidence of residual damage on removal of the anæsthetic. Levy was the first investigator to recognize that the not infrequent occurrence of cardiac arrest under chloroform was due to the anæsthetic agent. He demonstrated in cats that chloroform sensitized the heart muscle, making it susceptible to ventricular fibrillation in the presence of some exciting cause. Use of adrenalin to control bleeding, or surgical stimuli during periods of light anæsthesia such as induction or reawakening, he found would result in ventricular fibrillation. More recently Meek *et al.* have stated that a more likely cause of death under chloroform in humans is a direct toxic effect on the myocardium resulting in cardiac failure with no evidence of a terminal ventricular fibrillation.

Considerable work by laboratory and clinical investigators has been done on the arrhythmias developing under cyclopropane. Meek found in dogs that deep cyclopropane anæsthesia sensitized the heart to the effects of adrenalin to a greater degree than light cyclopropane. Atropine can only delay the appearance of the ventricular tachycardias after the use of adrenalin. Further studies would appear to demonstrate that no residual damage occurs as a result of the arrhythmias. Robbins and Baxter found that if anoxæmia is avoided by artificial respiration, if necessary, then arrhythmias did not develop even in deep cyclopropane anæsthesia. Vago-vagal reflexes produced by mechanical irritation of the trachea by intubation or tracheal aspiration are prone to result in ventricular extra-systoles and tachycardia; cyclopropane appears to be the greatest offender. Some degree of protection is offered by pre-anæsthetic atropinization to decrease vagal tone.

The problem of anæsthesia for thyro-cardiac patients requires special consideration inasmuch as the heart muscle is in a highly sensitized state as a result of the long-standing thyrotoxicosis. Lahey has reported ventricular fibrillation occurring when cyclopropane has been the anæsthetic; the use of an ethylene-ether combination has been recommended. Tovell feels that cyclopropane is safe providing that a small amount of ether is added as a protection for the heart.

Respiratory system.—Depression of respiration during anæsthesia is a hazard that is continuously confronting the anæsthetist due to the deleterious effects it may have on the oxygen supply and the carbon-dioxide elimination. Respiratory depression is not uncommon with cyclopropane since it lacks the irritants

of ether. Due to the high oxygen usually administered with cyclopropane, there is little danger of hypoxia developing but the impairment of carbon dioxide elimination may be a source of danger to the patient, as previously discussed.

The respiratory depressant action of pentothal sodium constitutes a definite hazard when administered to patients with advanced respiratory tract disease or respiratory distress, and should be avoided. Respiratory failure under pentothal sodium has been studied extensively by Beecher and Moyer. They found with prolonged pentothal anæsthesia a gradual retention of carbon dioxide and a decline in the arterial oxygen saturation. The normal sensitivity of the respiratory centre to carbon dioxide is lost; narcotic concentration (over 10%) may actually be found. Hypoxia now becomes the stimulus to respiration, acting through the carotid body. In the presence of this high carbon dioxide and low oxygen, small supplementary doses of pentothal for maintenance may produce a far greater effect than normal, resulting even in cardio-respiratory collapse. Apnœa results if oxygen is administered to these patients since the sole remaining drive to respiration—hypoxia—is removed. Maintenance of adequate tidal volume and avoidance of hypoxia must be assured in any prolonged pentothal anæsthesia.

The overactivity of the para-sympathetic nervous system under pentothal sodium resulting in laryngospasm has been studied in the cat by Burstein and Rovenstine. Pre-anæsthetic atropinization was found to block effectively this vagal overactivity. They believed that a similar basis exists in humans for the laryngospasm seen in humans under pentothal sodium.

The presence or absence of hazards in the use of ether for anæsthesia in cases of pulmonary tuberculosis appears to be the source of some disagreement among anæsthetists. Beecher and Adams maintain that ether, properly given in a closed system, is the safest anæsthetic; evidence is lacking that ether, more than any other agent, in any way contributes to the spread of the pulmonary tuberculosis.

The administration of clinically effective doses of avertin is followed by marked respiratory depression. The patient must be constantly observed to guard against upper respiratory obstruction being super-imposed on the respiratory depression.

Convulsions in anæsthesia.—The occurrence of convulsions under general anæsthesia in otherwise normal individuals is an unsatisfactorily explained phenomenon. However with the administration of 100% oxygen, withdrawal of the agent, and the prompt intravenous injection of a soluble barbiturate, there is generally a successful termination of the convulsion with no residual damage. Convulsions may occur under any general anæsthetic and have been attributed to a variety of causes. Rosenow and Tovell have demonstrated the presence of a neurotropic streptococcus as a causative factor in patients who have had convulsions. Waters has claimed that carbon dioxide retention is usually a feature of the anæsthetic course during which convulsions have occurred. In an analysis of 12 cases, Ray and Marshall have concluded that hypoxia is a precipatating factor. Bourne believes that in convulsions under ether, hæmoconcentration may be a causative factor.

The basis for any convulsion is probably the result of a combination of factors, but the observance of such basic principles of inhalation anæsthesia as avoidance of hypoxia and carbon dioxide retention should result in an even further lowering of the incidence of convulsions.

Anæsthesia in shock.—The administration of anæsthesia to patients in a state of shock certainly is "adding insult to injury" but is often unavoidable. The choice of agent then becomes of vital importance. Cyclopropane with its high oxygen percentage is recommended by some. Skilful administration of pentothal and nitrous oxide may be advantageous due to the relative non-toxicity of this combination. Experimentally, it has been shown that dogs die more readily from hæmorrhage under spinal than under ether anæsthesia. Beecher, in a review of the whole problem, has concluded that ether is the choice of agent if general anæsthesia must be used.

Explosive hazard.—The administration of any explosive inhalation agent constitutes, potentially, a serious hazard. Only chloroform and trilene are non-explosive and non-inflammable. Nitrous oxide is non-explosive but supports combustion. Unless precautionary measures are taken, there are many potential sources of ignition in the operating theatre. The various

forms of electrical equipment—cautery, x-ray units, suction and blower apparatus, and wall plugs, switches, and electrostatic discharges are the two major sources of ignition. Greene has reported in an analysis of 230 fires and explosions that all were preventable by the use of measures known at the time. The seriousness of the explosive hazard cannot be too greatly stressed. Here, as nowhere else, one should learn from the misfortunes of others. It is the duty of anæsthetists and hospital authorities to adopt all known precautions against the occurrence of explosions and fires in the operating rooms.

Regional anæsthesia.—The "local" anæsthetic drugs have provided surgeons and anæsthetists with a relatively controllable anæsthetic agent. The specificity of the action of these drugs on nervous tissue reduces to a minimum their systemic effects if administered in properly controlled dosage. The relative toxicity of the individual agents is affected by such factors as site of injection, amount of drug used, concentration of solution, and presence or absence of a vasoconstrictor. Inasmuch as the liver is the site of destruction of all local anæsthetics, the speed with which it can detoxify the drug is also of vital importance in determining the toxicity. The appearance of restlessness, nausea, vomiting, and tremors in many patients after local anæsthesia, may be evidence of central nervous system irritation. This may progress to convulsions, and possible death if treatment is not promptly instituted. The administration of oxygen and an intravenous barbiturate will successfully control such central irritation. All local anæsthetic agents such as cocaine, procaine, metycaine, pontocaine, or nupercaine, may produce evidence of central nervous system irritation. These may occur if the rate of injection or absorption from the site of injection exceeds the rate of destruction by the liver.

Cardiocirculatory effects are most common following cocaine, resulting in tachycardia and vasoconstriction of central origin. If the dose of cocaine is large or absorption rapid, cardiac collapse results. Acute cardiac arrest is rare, being totally unrelated to the dose of cocaine used. Toxic symptoms following procaine, metycaine or pontocaine less commonly manifest themselves in the cardiovascular system. Nupercaine, if administered in large doses, may result in depression of cardiac function leading to cardiac arrest. Considerable evidence, clinical and experimental, has accumulated showing that procaine is the safest of all the local anæsthetic drugs.

Spinal anæsthesia.—By far the most hazardous type of regional anæsthetic techniques is spinal anæsthesia, the hazard increasing greatly with increasing height of anæsthesia. The choice of agent is subjected to the same considerations of toxicity as discussed above. Howard Jones has stressed the importance of reducing the concentration of the drug used in toxic or debilitated patients since their reaction to any given strength of drug is much greater than that of the normal individual.

The outstanding hazard of spinal anæsthesia is its adverse effect on the cardiovascular system. The consistency and degree of the fall in the blood pressure increases with increasing height of anæsthesia. Papper, Bradley, and Rovenstine have critically reviewed the entire problem and have concluded that the induced changes are the result of a post-arteriolar stagnation of blood. The maintenance of the diastolic pressure, lowered cardiac out-put, increased arterio-venous oxygen difference, and increased circulation time are all evidence of impaired venous return to the heart. The prophylactic use of ephedrine to counteract these changes is recommended. Bieter maintains that ephedrine is less efficient if given after these cardiocirculatory changes have occurred. The incidence of the fall in blood pressure following spinal anæsthesia has been estimated at 70%.

CoTui and Burstein have presented evidence of the instability of the circulatory system under spinal block. Changes in position should be made with the utmost care. Hæmorrhage is poorly tolerated by animals under spinal anæsthesia, particularly high spinal anæsthesia where practically the entire compensatory mechanism is blocked. Furthermore, Papper *et al.* were able to show that the fall in blood pressure with high spinal anæsthesia (above D-6) is much greater and more common where operation is performed than when it has not been done. The need for gentle handling of tissues is clearly indicated.

Undue spread of the anæsthetic with increasing paralysis of the intercostal muscles is a grave hazard if unattended by prompt and

adequate therapy. The greatly diminished respiratory exchange may result in a serious degree of hypoxia with a further depression of the blood pressure. The inhalation of carbon dioxide for stimulation of respiration is contra-indicated since it results in a further lowering of the blood pressure due to peripheral vaso-dilatation. The normal reaction to carbon dioxide of vaso-constriction with elevation of the blood pressure, mediated through the carotid body, is prevented by the blockage of the vaso-constrictor fibres.

Clinical neurologic sequelæ of spinal anæsthesia are a rare but serious hazard. A wide variety of lesions have been reported, ranging from headache to permanent paresis. Nicholson and Eversole have shown that pooling of a concentrated solution of an anæsthetic agent in the sacral portion of the sub-arachnoid space is prone to produce at least transient nerve damage. It is not considered advisable to administer a spinal to any patient who shows evidence of pre-existing central nervous system disease. The anæsthetic agent acts as an irritant that may aggravate an otherwise quiescent lesion. The occurrence of these neurological lesions of a remote or immediate nature is rare, and should not constitute a contra-indication to spinal anæsthesia except as noted above.

The incidence of headache following spinal anæsthesia (and lumbar puncture for diagnostic purposes) has been reported as varying from 0.1 to 83%. Lemmon has reported an incidence of 3% following continuous spinal. The etiology is uncertain. Though usually of a mild self-limiting nature, it nevertheless constitutes a real hazard for which there is no special prophylaxis.

Curare.—This recently introduced drug possesses relatively few hazards. Respiratory depression has been reported by many anæsthetists. Curare should never be administered if the facilities are not readily at hand for performing efficient artificial respiration. If curare is administered during ether anæsthesia, the dose must be reduced to one-third of that commonly used with cyclopropane. When used by experienced anæsthetists, with facilities for maintenance of an adequate respiratory exchange if the need arises, curare appears to be one of safest drugs we have.

SUMMARY

An attempt has been made to present some of the hazards and untoward side-effects, particularly those having a pharmacological and physiological basis, that may be encountered in the use of the common anæsthetic agents and techniques.

Avoidance of many of these hazards lies mainly in the careful choice of agents or combinations of agents and their skillful administration. Despite the introduction of many potentially safer agents, the skill and knowledge of the anæsthetist is still a vital factor.

NOTE.—An extensive bibliography has been prepared and will appear in the reprints supplied to the author.

RÉSUMÉ

Revue des principaux dangers que présente l'anesthésie, et rappel de leurs fondements physiologiques et pharmacologiques. La prévention des accidents au cours de l'anesthésia dépend, tout autant que du choix des anesthésiques, de leur habile administration. En dépit de l'apparition de nouveaux produits d'une grande sécurité, l'art et la science de l'anesthésiste restent des facteurs de premier plan. PAUL DE BELLEFEUILLE

A NEW SYNTHETIC ANTI-HISTAMINE SUBSTANCE DERIVED FROM PHENOTHIAZINE (Phenergan, 3,277 R.P.)*

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THE name phenergan (3,277 R.P.) has been given to the N-dimethylaminopropyl derivative of phenothiazine. The anti-histamine properties of this synthetic substance were discovered in France in 1946[1] and have been intensively studied there since then.[2, 3]

Historical survey.—The first synthetic substance reported to possess antihistamine properties was described by Fourneau and Bovet in 1933[4] and later by Staub and Bovet in 1937.[5] This was 929 F. or thymoxyethyldimethylamine. In 1937, Bovet and Staub[6] also described 1,571 F. a derivative of ethylene diamine. Both these substances showed only moderate activity and were too toxic for therapeutic administration.

In 1942 Halpern[7] showed that N-dimethylaminoethyl-N-benzylaniline (antergan of 2,339 R.P.) was more powerful and less toxic than the earlier products and the first clinical trials were attemped.

During the years which followed, various modifications of Halpern's drug were produced, such as neoantergan (Bovet and Walthert, 1944[8]), benadryl (Loew, Kaiser and Moore, 1945[9]) and pyribenzamine (Mayer, Huttrer and Scholtz, 1945[10]). Numerous comparative

* From the Clinique Médicale Propédeutique, Hôpital Broussais, Paris (Professeur Pasteur Vallery-Radot).

studies have been made, notably by Code[11] and by Feinberg,[12] as well as the many studies on the experimental activity of each substance separately.[13, 14, 15]

In 1946, Halpern and Ducrot[1] showed that certain phenothiazine derivatives possessed anti-histamine properties which were in many respects more powerful than those of any previously described substance. A series of experiments was made to study the activity of this new group of substances, particularly N-dimenthylamino-2-propyl-1-phenothiazine, known as 3,277 R.P. or phenergan.

Experimental findings.—Phenergan possesses the same well-established properties as the other anti-histamines, but quantitatively its action is much more powerful in many respects. Its antagonism to histamine is well shown in experiments on the smooth muscle of the bronchi, intestine and uterus and by its inhibition of the triple response of Lewis and of the histamine effect on the blood-pressure.[7, 9, 24, 25] The degree of protection against histamine conferred by phenergan is much greater than that given by any other anti-histamine substance. In guinea-pigs, for example, a dose of phenergan of 20 mgm. per kilo. protects the animal against 1,500 times the lethal dose of histamine.[16]

The anti-anaphylactic effect is equally striking. Phenergan counteracts anaphylactic shock and prevents the Prausnitz-Küstner reaction. This action is much greater than that of the other anti-histamine substance. The duration of action against experimental asthma in the guinea-pig is 3 times as long as the action of antergan or neoantergan under the same conditions. Phenergan, like the other anti-histamine substances, cannot however counteract the effect of histamine on the secretions. The animals given large doses of histamine after the administration of phenergan show no immediate signs of histamine shock, but a considerable gastric hypersecretion is produced. This probably explains the constant appearance of gastric ulcerations under such conditions.[15]

Last and Loew (1947[16]) had already demonstrated by Menkin's method that benadryl and neo-antergan counteract the local increase of capillary permeability induced by histamine. By noting the penetration of fluorescein into the anterior chamber of the eye[19] and the diffusion of dyes into peritoneal exudates,[20] it has been shown that phenergan powerfully opposes the increase of capillary permeability produced by histamine and various other substances.

Two special effects of phenergan, possibly due to its more powerful action on capillary permeability, have been investigated. (*a*) Phenergan prevents the appearance of the acute pulmonary œdema which is induced in unprotected animals by intravenous epinephrine[21, 25] or by certain poison-gases, such as chloropicrin or phosgene.[22] (*b*) Phenergan prevents the induction of experimental orthostatic albuminuria in rabbits.[23]

CLINICAL STUDIES

Methods.—Phenergan was administered orally in the form of 25 mgm. tablets. The usual dose varied between 25 and 100 mgm. daily, but in certain cases, up to 200 mgm. were given. Blood counts were carried out before and every 2 weeks during treatment. Allergic, clinical and skin-tests were carried out before and during treatment whenever possible.

Results.—(*a*) *Serum sickness*: 7 cases of serum sickness were treated. In each case the skin manifestations such as pruritus and urticaria, disappeared within from 30 minutes to 3 hours after administration of the drug. Joint pains were present in 4 patients and disappeared under treatment in only 2 of the cases. The fever was scarcely influenced.

(*b*) *Urticaria*: 123 cases of urticaria of varying origin have been treated. In only 12 of these cases could an allergic basis be demonstrated by clinical and cutaneous tests; 108 cases (87.8%) showed immediate improvement. The pruritus was the first symptom to disappear, then the skin reaction tended to diminish and had often entirely disappeared after a few hours. Of the remaining 15 patients, 6 showed signs of intolerance and were unable to continue treatment, while 9 cases were little, or not at all, benefited by the drug. All the cases of definitely allergic origin improved under treatment, but among the successes were several cases in which there was no reason to suspect any such cause. About 60% of the successful cases had previously failed to respond to neo-antergan.

(*c*) *Angio-neurotic œdema*: Out of 19 cases of angio-neurotic œdema, only 3 were not improved by treatment (84% of favourable results). Two of the successful cases were patients in whom œdema of the lips had persisted in between the acute exacerbations. Apart from these cases of true angio-neurotic œdema, there were 2 patients who suffered a local œdema after the slightest pressure to any part of the body. These cases were uninfluenced by phenergan.

(*d*) *Pruritus*: Out of 18 cases of prurigo from varying causes, 6 were uninfluenced by phenergan, but 12 were vastly improved, the

pruritus ceasing even when the visible lesions persisted unchanged. Included in these 12 *are* several cases of pruritus due to scabies.

(*e*) *Eczema*: None of our 17 cases of chronic eczema was completely cured by treatment. In a few cases slight relief was obtained, especially from the subjective symptoms such as pruritus. Out of 22 cases of acute eczema and contact dermatitis, 3 showed rapid improvement which ended in complete cure. The rest of the cases improved only after a much longer period and it is difficult to assess the influence of phenergan on these results. Three cases of arsenical and one case of gold erythrodermia showed no real improvement on treatment with phenergan.

(*f*) *Allergic purpura*: One case of allergic purpura, in which new crops of petechiæ appeared daily for three weeks before treatment, was treated with phenergan. Administration of the drug was followed by immediate cessation of fresh eruptions.

(*g*) *Hay fever*: 142 cases of hay fever were treated between the spring of 1947 and that of 1948; 98 cases (69%) showed complete disappearance of all signs and symptoms; 36 cases were only partially relieved; the sneezing ceased but the nasal congestion remained more or less the same; 8 cases were completely unchanged by treatment but most of these were unable to tolerate full doses of the drug.

It should be noted that, in the majority of cases, 25 mgm. of the drug daily suppressed sneezing, but a dose 4 to 6 times as great is usually necessary to give complete relief from all the symptoms. For a few patients, however, 6 to 12 mgm. daily sufficed to suppress all symptoms.

At a recent meeting of the French Society of Allergy (June 15, 1948) Pasteur Vallery-Radot, Blamoutier and B. N. Halpern have reported a series of 200 cases of hay fever treated with phenergan with a dose of 25 to 50 mgm. daily; they reported 86 per 100 of excellent results with relief of all symptoms; only in 14 per 100 the improvement was incomplete.

TABLE OF RESULTS

Condition	Total No. treated	Cure		Improvement		Failure	
		Actual No.	Percentage	Actual No.	Percentage	Actual No.	Percentage
(*a*) Serum sickness	7			7	100.0	0	0
(*b*) Urticaria							
(*1*) Definitely allergic	12	<———		12 ——>	100.0	0	
(*2*) Etiology unknown	111	<———		96 ——>	86.5	15	13.5
Total	123			108	87.8	15	12.2
(*c*) Angioneurotic œdema	19	<———		16 ——>	84.0	3	16.0
Pressure œdema	2	0		0		2	
(*d*) Pruritus	18	<———		12 ——>	67.0	6	33.0
(*e*) Eczema							
(*1*) Acute and contact	22			3	13.6	19	86.4
Chronic	17	0		0	0.0	17	100.0
Total	39			3	7.7	36	92.3
(*2*) Gold and arsenic erythrodermia	3	0		0		3	
(*f*) Allergic purpura	1	1					
(*g*) Hay fever	142	98	69	36	25.3	8	5.7
(*h*) Asthma							
(*1*) Definitely allergic	9	<———		9 ——>	100.0	0	
(*2*) Etiology unknown	63	0		21	33.3	42	66.6
Total	72			30	41.7	42	58.3
(*i*) Spasmodic cough	9	0		0		9	100.0
(*j*) Migraine	20			6	30.0	14	70.0

(*k*) Other conditions—Acute and subacute rheumatism, tuberculous allergy, acute glomerulo-nephritis—uninfluenced

(*h*) *Asthma*: 72 cases of asthma were treated, but an allergic basis could only be demonstrated in 9 of them. In all these 9 cases, considerable relief or even complete disappearance of the asthma was obtained. In 21 other cases without any appreciable allergic origin, there was a certain degree of improvement. In the remaining 42 cases, the results were completely negative.

(*i*) *Spasmodic cough*: 9 cases of spasmodic cough were entirely uninfluenced by treatment.

(*j*) *Migraine*: Out of 20 cases of migraine, 6 were improved by phenergan. It was impossible to determine an allergic basis for any of these 6 cases.

(*k*) Phenergan appeared to have no effect on chronic or sub-acute rheumatism or on acute glomerulo-nephritis. It did not seem to influence the allergic manifestations of tuberculosis, or the intradermal tuberculin test. This latter observation is in contrast to its action on allergic skin tests for other diseases. In such cases, whenever skin tests were found to be positive before treatment, immediately after adequate dosage with phenergan they became transitorily negative.

TOLERANCE AND SIDE-EFFECTS

The drug was usually well tolerated. In particular, it did not provoke the digestive upsets which often occur with the other anti-histamine substances. The blood-picture was only affected in 2 cases, a slight neutropenia being observed. Administration of the drug was immediately stopped. The only important side-effects were of nervous origin. About 25% of the patients showed a slight degree of drowsiness accompanied by vertigo and instability when standing upright and by sensations of drunkenness. Occasionally there was also a slight decrease in intellectual power. In rare cases insomnia occurred rather than drowsiness.

These troubles, which are impossible to predict and which may occur even with feeble doses, constitute the most serious disadvantage to the use of phenergan. Usually they seem to be partially neutralized by the simultaneous administration of benzedrine. Moreover, the nervous troubles almost always disappear within a few days, even if treatment is continued with the same dose. The drowsiness may be neutralized if phenergan is given at night. This effect wears off before morning but the true action of the drug continues throughout the following day.

DISCUSSION

The experimental and clinical results described above lead us to consider phenergan as a powerful anti-allergic substance. However, several conditions in which no allergic cause could be demonstrated were influenced by the drug. These were: (*a*) experimental syndromes such as acute pulmonary œdema due to epinephrine or poison-gas; orthostatic albuminuria; (*b*) clinical cases of migraine, certain skin conditions and some asthmas. On the other hand, a certain number of definitely allergic conditions were not benefited by the drug, for example, all chronic and most acute cases of eczema.

In consequence, the problem of the mechanism of action of phenergan still has to be elucidated. The first theory was that histamine is responsible for non-allergic syndromes influenced by phenergan. Up to the present there has been no serious proof of this. The experimental work described above suggests that phenergan acts on capillary permeability. In this connection, it is very striking that most of the pathological conditions controlled by phenergan are characterized by serious extravasation through the capillary wall — for example, experimental acute pulmonary œdema and orthostatic albuminuria, urticaria and even, perhaps, migraine.

SUMMARY

1. Experimental work on phenergan, a phenothiazine derivative which is a powerful new anti-histamine substance, is described. Apart from its action on allergic conditions, it prevents the production of experimental acute pulmonary œdema and orthostatic albuminuria.

2. Clinical trials with this substance proved it to be of value against serum sickness, urticaria, angioneurotic œdema, hay-fever and allergic purpura. Less success attended the use of phenergan in cases of asthma, migraine and various types of pruritus, with almost none against the eczemas.

3. The mechanism of action of phenergan is discussed. The experimental and clinical evidence are in favour of its action on capillary permeability.

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ARE THERE NUTRITIONAL PROBLEMS IN CANADA?*

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THERE are so many nutritional problems in Canada that it is possible to discuss only a few of them in any detail. These nutritional problems may be considered in two large groups: (1) problems of a general nature; and (2) problems of a specific nature. The general problems will require long term programs for solution, the specific problems, such as the extent of vitamin deficiencies in the population, are of immediate concern to every practitioner who is interested in the best possible prognosis for his patient.

Malnutrition has been compared with an iceberg in which only a small amount appears obviously on the surface, and a much larger amount lurks hidden. In this respect it is no different than other diseases, in which the problem is indicated only to a very limited extent by statistics of mortality rates for example.

TABLE I.

NUMBER OF DEATHS ASCRIBED DIRECTLY TO NUTRITIONAL DEFICIENCY DISEASES IN CANADA, 1937 TO 1946 INCLUSIVE.

	Scurvy	*Beriberi*	*Pellagra*	*Rickets*	*Osteomalacia*
1937	7	1	2	54	5
1938	11	1	2	61	4
1939	11	2	6	49	4
1940	6	1	3	66	5
1941	3	4	4	48	5
1942	2	3	2	45	1
1943	6	1	4	37	1
1944	4	1	3	34	1
1945	5	2	5	22	3
1946	1	3	3	29	4

To the basic figures in Table I must be added many other types of malnutrition which do not reach the records as the primary cause of death, although they may be serious in various ways. These other types include iodine deficiency and simple goitre, riboflavin deficiency and lesions in tongue and eyes, iron deficiency anæmia, underweight and thinness, which give poor reserves, overweight and obesity with their proved danger to health and longevity, protein deficiency with many concomitant effects in relation to wound healing, immunity, etc., and various other vitamin deficiencies.

All of these milder types of malnutrition have been found in Canada, from British Columbia to Nova Scotia and among all economic levels. An average figure for their incidence is not yet possible, but nutrition surveys are gradually completing the picture. Surveys have been carried out by the Department of National Health and Welfare using methods already described, and some of the results are presented in Table II. These results are not presented here for detailed discussion, since this would take too long, but only to emphasize that nutritional problems are found all over Canada, and that they vary in their extent from one area to another, even in the same Province. To these results of ours should be added the medical nutritional surveys by McHenry and his colleagues in Toronto, and less complete studies by others. I do not include the evidence obtained by various people all over Canada as a result of food studies alone, because it is not possible to

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interpret such dietary surveys in terms of actual or potential malnutrition with the same degree of assurance that is possible when the patient is seen and his blood tested, as in our nutritional surveys.

RICKETS STILL A PROBLEM

It can be seen in Table II that *rickets* is still a problem in Canada. Not the florid rickets of the first 6 months of life, but rickets nonetheless. This was shown by the mortality figures in Table I, but it is even more emphasized in Table II. In some areas one of 3 children, in other areas one out of 20 show permanent bony deformities that have been described as rachitic. This is in accord with our dietary findings that 37 to 91% of all children, even in the first year of life, were getting no source of vitamin D.

Fish liver oil, or some other source of vitamin D is one of the most specific preventive measures available in medicine. This lack of vitamin D is not our only nutritional problem in Canada, but it shows a neglect that makes it one of our most important problems right now. A lack of food, leading to underweight and a lack of subcutaneous tissue which we combine in the term "thinness" is a constant finding in a percentage of people, especially children.

A lack of riboflavin is often diagnosed, although there are dramatic differences in its incidence in different parts of Canada.

Vitamin deficiencies, other than of vitamin D, and riboflavin, are rarely diagnosed. This is especially noteworthy in the case of the B vitamins, niacin and thiamine. Pellagra, as such, is very rare in Canada, and evidence of even mild deficiencies are not nearly as common as reported in the United States. This fact is important in relation to the controversy on adding three B vitamins to flour and bread, a practice started in the United States during the war, but which is forbidden in Canada. Convincing evidence for adding niacin or thiamine to flour or bread in Canada is not available, and the third one—riboflavin—is readily obtainable from milk, whether directly or added to bread.

TABLE II.

SOME OF THE NUTRITIONAL DEFICIENCIES FOUND BY DIRECT SURVEY IN DIFFERENT AREAS IN CANADA. (TOTAL NUMBER OF PEOPLE SURVEYED IS OVER 6,000).

FIGURES GIVE PERCENTAGE OF THOSE SURVEYED IN EACH GROUP

Definite or probable deficiency	*British Columbia (children only)*	*One area in Alberta (all ages)*	*Saskatchewan (children only)*	*Two areas in Saskatchewan (all ages)*		*One area in Ontario (all ages)*	*Two areas in Quebec (children only)*		*One area in Nova Scotia (all ages)*
	%	%	%	%	%	%	%	%	%
Riboflavin	20.0	5.0	22.0	3.0	1	0.6	23.0	10	8.0
Thinness	14.0	2.0	11.0	4.0	4	12.0	13.0	14	4.0
Past rickets	11.0	30.0	12.0	19.0	22	4.0	16.0	10	13.0
Low hæmoglobin	8.0	5.0	3.0	8.0	14	11.0	5.0	4	6.0
Vitamin A deficiency	7.0	0.0	9.0	1.0	0	8.0	3.0	2	2.0
Ascorbic acid deficiency	0.7	0.0	3.0	0.6	3	0.0	0.6	1	5.0
Protein deficiency	0.7	0.0	0.1	0.0	0	0.0	0.0	0	0.9
Niacin deficiency	0.0	0.0	0.0	0.6	0	0.0	0.0	0	0.6
Thiamine deficiency	..	0.0	..	0.0	0	0.0	..	..	0.0

Carious teeth, poor posture, obesity, simple goitre and other problems were also encountered.

OVERWEIGHT A PROBLEM

Overweight is a problem of great importance. It shortens life, decreases efficiency at work, increases liability to many diseases: cardiovascular disease is more common; 70% of diabetics are overweight; gall stones and biliary tract infections are more common; hernia, diverticulitis, bronchitis and emphysema are all more common or more difficult to treat in stout subjects; pneumonia and other infections are more dangerous; and the mortality rate in surgery rises. The exact extent of overweight in Canada is not easy to determine from our results so far, partly because the standard to use is in some doubt, but rarely have we encountered "overweight" in less than 10% of the adults in a given area.

Even among adults there is a problem of underweight among a great many people. Sometimes there are just as many underweight as overweight people in an area. In both cases plus or minus 10% from the usual medico-actuarial tables is allowed as normal.

GENERAL PROBLEMS IN NUTRITION

Those are some of the specific nutritional problems in Canada. In concluding I wish to refer to a couple of more general problems. The first is the lack of adequate nutrition training in medical schools. Nutrition gets a mention in several different courses in medical training, and this is the proper way to handle it. But there is a need in every medical school for a nutritional specialist whose duty it is to see that the various bits and pieces fit together, so that the graduate sees the subject as a whole as well as in its various applications. In only a few American universities, and no Canadian, has such a senior position been established. This is one of the real problems for the future.

A second general nutritional problem, partly resulting from the lack of training mentioned above, is the fact that most hospital diets (based on the few studies available) do not even meet the minimum needs of patients, let alone providing the hyperalimentation that can contribute to early convalescence and can reduce complications and mortality. Every hospital food service should be so well staffed, equipped and financed that it can make the very real therapeutic contribution of which it is capable. Very few are doing it at the present time.

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Infrared photography has been used to study the superficial venous pattern which may be altered by any condition which affects the circulation; 100 patients with breast complaints entering a dispensary from a cancer detection clinic were studied with the purpose of ascertaining (1) whether the pattern of the superficial veins of the thorax would be altered or disturbed by a tumour within the breast. (2) Whether the tumours were malignant or benign, and (3) whether infrared photography might be of practical assistance in the diagnosis of cancer. Infrared Photographic Study of the Superficial veins of the Thorax in Relation to Breast Tumours. A Preliminary Report. Massopust, Leo C., *Surg., Gyn. & Obst.*, **86**: 54, 1948.

THE USES OF STREPTOMYCIN IN UROLOGY*

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PART II.—THE ORAL USE OF STREPTOMYCIN AND SULFATHALIDINE IN URETERO-INTESTINAL TRANSPLANTS

IT has been shown by Morton[2] that by the oral administration of streptomycin, the floral content of the bowel can be reduced to an exceedingly low level, and in many cases after administration no growth was obtained on culture of the stool (see Fig. 1). To achieve this, preparation of the bowel is commenced one week prior to operation by giving sulfathalidine orally. In addition streptomycin, 4 grams daily, is given orally (in orange juice) commencing 3 days prior to operation.

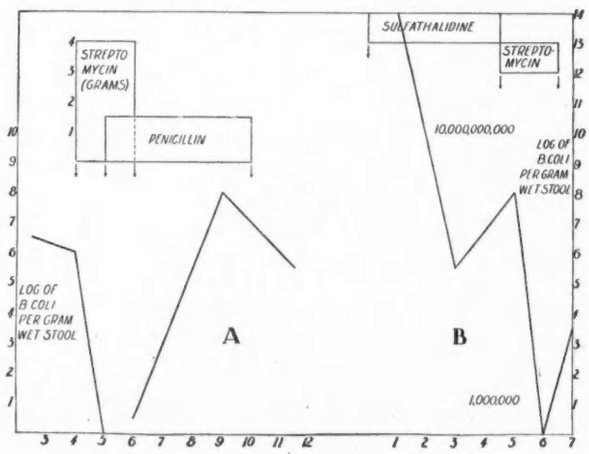

Fig. 1.—(*a*) The effect of streptomycin orally on *B. coli* in the stool. Note the return of growth when streptomycin orally is discontinued and penicillin is given intramuscularly (modified after Morton[2]). (*b*) Initial sharp decline in growth of *B. coli* in bowel, by using sulfathalidine orally; and the further marked decrease to zero when streptomycin orally is added.

We have used this technique with slight variations, in 3 cases of uretero-intestinal transplants in which 4 operations were done. In our cases the initial preparation was with sulfathalidine, followed by streptomycin, 2 grams daily, orally, for 3 days prior to operation and continued for 2 days postoperatively. Cultures were taken from the lumen of the bowel at the time of opening the bowel. Three cases are presented.

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CASE 1

G.B., aged 53 years. This patient was admitted to the Royal Victoria Hospital on August 1, 1946, complaining of frequency every hour, burning micturition, and terminal hæmaturia of six months' duration. He gave a history of having had the left kidney removed 33 years previously for tuberculosis. Physical examination was essentially normal, except for the genito-urinary organs. The penis, testes and epididymi were normal. On rectal examination the sphincter tone was normal. The prostate was found to be small, irregular and very firm. The prostatic fluid was loaded with pus cells and red blood cells. He was thought to have a tuberculous prostatitis and probably tuberculosis in the sole remaining kidney. A cystoscopic examination was done on August 7. The findings were as follows:

Filiforms and followers were passed with relative ease, although on a previous occasion the stricture in the bulbous portion of the urethra could not be passed. A French No. 21 cystoscope was then introduced. The trigone and ureteral orifices were normal in appearance. Clear urine was seen to come from the right ureteral orifice. The bladder wall elsewhere showed no evidence of tumour, ulceration, diverticulum or stone. Previous intravenous pyelogram series showed a double renal pelvis and a double ureter on the right, which fused in the lower third. Retrograde pyelograms were therefore not done.

Conclusion.—Double renal pelvis and double ureter, right, incomplete.

Repeated guinea pig inoculations and cultures of 24 hour specimens of urine carried out over a period of months, were entirely negative for tubercle bacilli. Repeated dilatations of the urethra and cystoscopic examinations were done at approximately monthly intervals. He was given intensive chemotherapy with sulfonamides, penicillin and streptomycin. During this 6 months period the bladder capacity gradually decreased in size, until the patient was finally voiding 42 times in 24 hours. It was felt that this patient probably had an interstitial carcinoma of the bladder, in spite of the fact that there was no evidence of this cystoscopically, and that he had had a previous nephrectomy for tuberculosis. A ureteral-intestinal transplant and total cystectomy was therefore recommended. The ureteral intestinal transplant was carried out on the right side extraperitoneally on September 22. The bowel had been prepared preoperatively with sulfathalidine and streptomycin. *A culture of the bowel content taken at the time of operation showed "no growth" of organisms.*

Postoperatively the patient had a most interesting and complicated course. No urine was obtained per rectum for 6 days postoperatively. The wound continued to heal per primam and there was no urinary leakage. The patient became more and more œdematous and the non-protein nitrogen rose to 109 mgm. % and the creatinine to 9.0 mgm. %. It was decided to open up the wound and insert a T tube in the ureter, if the patient did not put out urine per rectum within the next 24 hours. That night he passed 40 ounces of urine, and from there on his convalescence was uneventful; the œdema entirely disappearing and the non-protein nitrogen returning to 29.5 mgm. %. He was discharged from the hospital on the 20th postoperative day.

Three days later the patient was readmitted to the hospital in coma, with a marked acidosis. His wife stated that he had been having 19 to 20 bowel movements per day since discharge from the hospital. On admission his CO_2 combining power was 18.7 vol. %. His blood sugar was 97 mgm. % and the chlorides were 428 mgm. %. The non-protein nitrogen was 46.0 mgm. %. The most interesting feature however, was that he had also been losing potassium, the blood level being down from a normal of 18-20 to 13.9 mgm. %. He was given 1/6 molar sodium lactate intravenously and potassium chloride and sodium bicarbonate by mouth. Within 48 hours his blood chemistry had returned to normal. The diarrhœa however continued. A detailed electrolyte study was made by the metabolism department and he was found to require 200 grains of sodium bicarbonate daily to maintain a normal acid-base equilibrium.

During the next 4 months the patient exhibited a very bizarre picture, a profuse diarrhœa, recurrent bouts of temperature but never any tenderness over the right kidney. On two occasions he was readmitted to the hospital for streptomycin therapy, both orally and intramuscularly. A study of the effect of streptomycin on his stool cultures is shown in Fig. 2.

This clearly demonstrates that although the patient had streptomycin-sensitive organisms, streptomycin orally did not appreciably alter the bacteriological count of the stool, because the repeated bowel evacuations were washing out the streptomycin before it had an opportunity to exert its effect. After another prolonged bout of diarrhœa, it was thought possible

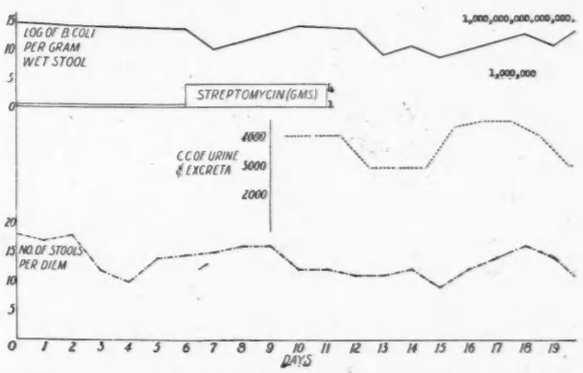

Fig. 2. (Case 1).—Indicating the negligible effect of streptomycin orally on *B. coli* in stool, in the presence of marked diarrhœa (modified after Morton[2]).

that this might be due to the destruction of some of the normal intestinal flora, by streptomycin, which flora were in fact essential. He was therefore given normal human fæces mixed with a disguising powder in a capsule, three times a day for three days. Following this, there was no change whatsoever in the clinical picture, the diarrhœa continuing unabated 19 to 20 times per diem.

Two weeks later the patient developed intense bladder spasms with the passage of blood clots. This continued, and he was readmitted to the hospital for re-evaluation and probable cystectomy. A cystoscopic examination at this time showed a definite tumour presenting on the left lateral wall of the bladder. It was therefore decided to do a cystectomy. Using Wishard's[3] quotation "We felt like the Scotch ferryman in a rowboat taking two ministers across the Loch Ness in a storm who said 'The wee one can pray, but the big one can man an oar' ".

On March 12, 1948, a total cystectomy was done, the prostate and seminal vesicles being removed *in toto* with the bladder. He had a carcinoma of the bladder (see Fig. 3).

Pathological report, March 17, 1948.

Bladder.—Specimen consists of a well-fixed urinary bladder which includes the prostate, seminal vesicles, and the severed distal ends of the ureters. Externally it is covered by a fibro-fatty tissue, a rubbery hard mass bulges posteriorly from the bladder wall. The bladder has been previously opened on its ventral aspect, revealing a markedly contracted cavity, which is 4 cm. in length in its longest diameter. On the posterior wall there is a round raised, irregularly nodular mass of rubbery, hard tissue, showing a central ulceration. Sections of the bladder reveals a large mass of rubbery, hard white homogeneous tissue, extending through the wall of the bladder from the area described above. The central portion is necrotic and presents a soft friable variegated red, gray and brown appearance. The seminal vesicles and prostate are grossly involved. The tumour extends in the wall of the bladder both inferiorly and posteriorly and superiorly, above to reach the fundus and below to reach the prostate. The remainder of the mucosa is thickened, highly rugose, œdematous and cobbled, in places it is covered with fibrinous exudate. The ureteral orifices cannot be defined.

Fig. 3. (Case 2).—Bladder removed at operation showing carcinoma solidum involving the entire bladder.

Microscopic examination.—Sections of the bladder show the whole of the wall of the organ to be extensively occupied by a growth of neoplastic epithelium, which completely replaces the mucosa and extends through the muscularis and largely destroys it, to reach the areolar tissue surrounding the bladder. The tumour is necrotic superficially and consists of a pleomorphic, anaplastic solid growth of epithelial cells which are oval to triangular in shape. Their cytoplasm is eosinophilic while the nuclei are large, vesicular, with distorted outlines, many have a hyperchromatic tendency, and show large red nucleoli. Mitotic figures are common. The prostate is not involved by tumour.

Anatomical summary.—Carcinoma solidum, urinary bladder.

His postoperative course was entirely uneventful. But now, the most interesting feature of the case developed. It was presumed that the cause of the diarrhœa was that the ureteral transplant was so situated as to be giving the patient a continuous high colonic irrigation. When the bladder, prostate and seminal vesicles were removed, the bowel evacuations dropped down to 6 or 7 a day, and have remained there ever since. The patient however still required sodium bicarbonate to maintain a normal CO_2 combining power. He was discharged from the hospital on the twenty-first postoperative day.

At the time of discharge his creatinine was 1.3 mgm. %; non-protein nitrogen 43.1 mgm. %; chlorides 337 mgm. %; CO_2 capacity 71.0 vol. %; total proteins 7.10 gm. %; albumen 4.42 gm. %; globulin 2.77 gm. %; serum sodium 325 mgm. %; serum potassium 17.26 mgm. %; serum calcium 10.8 mgm. %.

CASE 2

L.T., aged 52 years. This patient was admitted to Queen Mary Veterans' Hospital December 6, 1947. His illness began 7 years ago when in 1941 he was investigated at another hospital for frequency and hæmaturia. No special treatment was given. When overseas, he was discharged back to Canada in 1943 because of a recurrence of the symptoms. In April 1946, he was admitted to Q.M.V.H. for investigation. At that time he was complaining of urinary frequency every 20 minutes to 2 hours during the day time, and four times at night; burning at termination of urination; pain in the left side while working; loss of 15 pounds weight, and terminal hæmaturia. An intravenous pyelogram done April 25, 1946, showed poor excretion but nothing diagnostic. Urine cultures showed a micrococcus and diphtheroids. A diagnosis of prostatic hypertrophy and prostatitis was made and on May 3rd, the patient was discharged.

He was readmitted to Q.M.V.H. December 19, 1946, because of persistence of frequency and hæmaturia. He now had a day frequency of 20 to 30 times and a night frequency of 5 to 6 times. He also had some tenesmus and dysuria. Acid and alkaline serum phosphatase were 0.2 and 4.4 units respectively. Many urinalyses repeatedly showed red blood cells and white blood cells and a marked albuminuria up to 130 gm. %. On January 7, 1947, a transurethral resection was done. On the right lateral and left lateral floor of the bladder a papillary growth was seen and subsequent pathological sections revealed evidence of a malignant papilloma. On January 22, suprapubic resection of the bladder was done, and a large papillary growth measuring 15 x 10 x 7 cm. was removed. The bladder was resected and the area fulgurated. Subsequent pathological diagnosis of this specimen also showed papillary carcinoma. Following operation the patient had much difficulty with pain and burning for one month, but his wounds closed satisfactorily. He was given antibiotic therapy using sulfonamides, penicillin and streptomycin. By March 15, except for some frequency and hæmaturia he was well. On March 28, he was discharged from the hospital.

During 1947, the patient spent 3 months in a civilian hospital for suprapubic pain, frequency and dysuria. Seven weeks prior to this admission the patient was in a second civilian hospital for the same complaint. He was then four weeks in the convalescent hospital, where he passed small clots of blood, finally being readmitted to the Ste Anne's Hospital on November 24.

On this present admission he complained of continued suprapubic pain, described as a lot of mice biting inside and also of very slight pain while passing water, and marked frequency every five minutes. He wore a rubber bag during the day. His physical examination at this time revealed the prostate to be quite tender; otherwise, negative. Intravenous pyelograms were essentially normal. Urinalysis at this time showed 80 to 100 white blood cells per high power field and many red blood cells. Urine albumin was 35 mgm. %. On December 17, a bilateral ureteral transplant into the sigmoid colon was performed. The left ureter was transplanted about 20 cm. above the right ureter. For 6 days preoperatively this patient had sulfasuxidine, 1.5 gm. every 4 hours. In addition he had streptomycin, 0.25 gm. orally every 3 hours for 3 days preoperatively and for 2 days postoperatively (a total of 10 gm.). Postoperatively he was also given 12 gm. of streptomycin intramuscularly during a 12 day period. The bacteriological reports of the stool cultures and bowel cultures were as follows: December 12: Moderate growth of usual flora—(preoperatively). No enteric pathogens. December 17: Swab from bowel at operation: Very light growth of usual flora. December 20: Rectal tube drainage: Heavy growth of pseudomonas. Moderate growth of *E. coli* and usual flora.

His postoperative course was quite stormy and he never fully recovered. He ran a continuous fever and an elevated white count up to 17,000. The non-protein nitrogen gradually rose to 96 mgm. %. Subsequent intravenous pyelograms on January 19, revealed the concentration of the dye to be very subnormal. There was a moderate dilatation of the minor calyces but no gross dilatation of the pelvis. There was a small amount of dye present in both kidneys at 2 hours but none at 3 hours. He developed a marked jaundice. The Vandenberg was direct, negative in one minute. Bilirubin 1.7 mgm. %. No satisfactory explanation of this jaundice was found, and it subsequently cleared up. The abdominal incision was now completely healed. He continued to run a septic course in spite of extensive antibiotic therapy, gradually going down-hill and expiring January 24.

At autopsy, the findings were:

Right kidney weighs about 350 gr., measures 12.5 x 6 x 3 cm. The perirenal capsules strip easily to reveal a very pale granular surface. The organ has a "boiled" appearance, looks swollen, is soft and friable in consistency. Scattered over the surface are multiple small 0.3 cm. nodular cortical abscesses. On section the whole parenchyma is creamy-white in colour except for a few of the pyramids which are dark red in colour. The cortex is of uniform thickness throughout, measuring about 0.8 cm. in diameter. The medulla also is of normal size. Scattered throughout are small areas of petechial hæmorrhage and small abscess cavities similar to that described above. The pelvis and calyces appear slightly dilated. The lumen is filled with thick creamy pus.

Left Kidney weighs 300 gm., and measures 12 x 6 x 2.5 cm. The capsule and perirenal fat strips easily. Several large areas of hæmorrhagic extravasations of blood measuring about 3 cm. in diameter are seen in the region of the left suprarenal gland and also on the external surface of the kidney. The external surface of the kidney is also of creamy-pale colour similar to the right, but no cortical abscesses are seen. This kidney is more firm and is less friable in consistency than the right kidney. It does not appear as swollen. The cut surface has the same general appearance as the right kidney, but no abscess cavities are seen. However, projecting from the lumen of the pelvis, a small rubber catheter is seen. This catheter extends down the ureter to within 4 cm. of the ureteral orifice.

Ureters, bladder and prostate.—The right ureter is dilated especially at its distal end where its lumen measures 1.5 cm. in diameter and is filled with the same creamy pus as seen in the pelvis. The distal end of the ureter has been amputated 4 cm. from the bladder and the proximal end has been transplanted into the lower sigmoid about 18 cm. from the rectum. The intrasigmoid stump measures 1 cm. in length and is well epithelialized. The lumen is easily patent to a 0.2 cm. probe. There is some ulceration of the mucosa of the sigmoid at the base of the stump, otherwise no inflammatory reaction is seen. No evidence of leakage is present.

The left ureter has also been transplanted into the sigmoid, and is 20 cm. proximal to the right ureter.

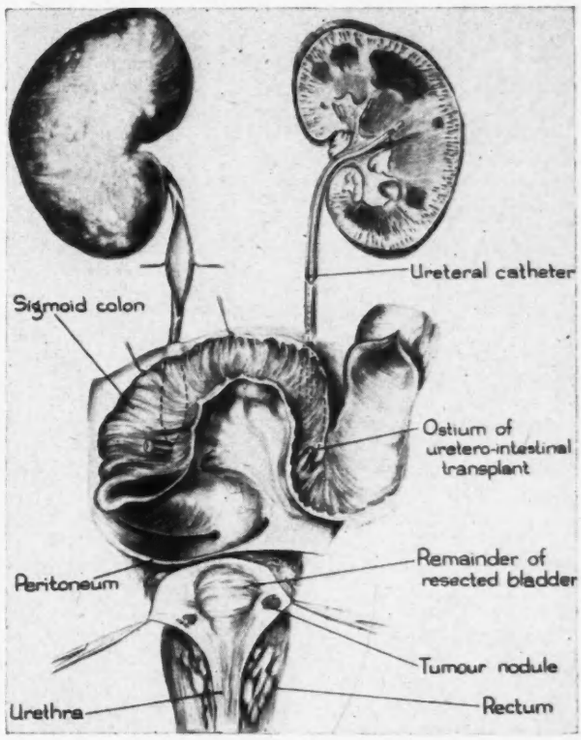

Fig. 4. (Case 2).—Autopsy specimen showing catheter *in situ* in left ureter. Note hydroureter on each side and the small size of the ureteral orifices in the sigmoid.

The intrasigmoid stump also measures 1 cm. in length and is well epithelialized. The lumen is easily probed with a 0.2 cm. probe, no ulceration is seen about this stump. No evidence of leakage is seen. The proximal portion of the ureter is also dilated to 1 cm. in diameter and filled with thick creamy pus similar to that seen in the pelvis of this kidney. The rubber catheter previously mentioned extends to within 4 cm. of the ureteral orifice. The catheter measures about 0.2 cm. in diameter and would not be able to pass through the orifice. In both instances the sigmoid at the site of the transplant has been sutured to the posterior peritoneum. No inflammatory reaction is seen.

The bladder was opened anteriorly. It is small and the lumen measures about 15 c.c. in volume. The wall is thickened by what appears to be fibrous tissue. On the mucosal surface there is a red papillary nodule measuring about 2 cm. in diameter and 0.3 cm. in height on the anterior aspect of the fundus. Also scattered throughout the fundal mucosa are a few red flecks similar to the tissue seen in this papillary formation. No large area of tumour formation is seen. The ureteral

orifices are patent and the ureteral stumps which are 4 cm. in length are well closed off.

The prostate is small, presents no gross evidence of obstruction. On section no gross lesions are seen. No glands are seen in the bladder region, nor is there any gross evidence of carcinomatous invasion of the perineum. One para-aortic gland was removed for section at the level of the kidney.

The whole picture is that of extensive pyo-ureters and pyonephrosis resulting from ascending infection up the ureters from the sigmoid colon.

Conclusion.—The loss of the catheter in the left ureter was a major complication. Strangely enough, at autopsy, the right kidney was more severely infected than the left. It is remarkable how small the ureteral orifices in the sigmoid have become as healing has become complete.

CASE 3

J.H., aged 26 years. This patient was admitted to Queen Mary Veterans' Hospital on August 8, 1947, for treatment of a chronic cystitis and sycosis barbæ. He first came under our care in 1945 at which time he was complaining of frequency every hour, for the past two years. He had been sent back from overseas because of this. There was no pain or burning on micturation, and no hæmaturia. He would pass about 50 c.c. of urine every hour. This greatly disturbed his rest, and he became thin, anxious and nervous. A detailed investigation in 1945 failed to reveal any abnormality other than a grossly inflamed bladder with some narrowing of the lower end of each ureter. The kidneys and pyelograms were entirely normal in appearance. He had in addition a sycosis barbæ involving the entire bearded area of the face. Repeated guinea pig inoculations and cultures of 24 hour specimens of urine failed to reveal any tubercle bacilli. Pyocyaneus and non-hæmolytic streptococci were found on urine culture.

Over a two-year period this patient was given intensive courses of the sulfonamides, penicillin, streptomycin, arsenicals, bladder dilatations under spinal anæsthetic, continuous bladder irrigations, etc. His condition became progressively worse. He was now voiding every ½ hour, had become exceedingly thin, and developed a depressive type of psychosis. He was admitted to the hospital on August 8, 1947 for transplantations of both ureters into the bowel. The psychiatrist felt that he should be given insulin shock therapy first. This was done.

The right ureter was transplanted into the sigmoid (extraperitoneally) on December 9, 1947. He was given sulfathalidine 1 gm. orally every 4 hours for 3 days preoperatively and 1 day postoperatively. In addition he was given 0.25 gm. of streptomycin orally every 3 hours for 3 days preoperatively and 3 days postoperatively. The bacteriological report was as follows: *Swab from bowel at operation*: Very light growth of usual flora. Recovery was uneventful and the patient was discharged to his home December 23.

He was readmitted January 3, 1948 and given exactly the same preoperative preparation of sulfathalidine and streptomycin as he had before the right ureter was operated upon. The left ureter was transplanted to the sigmoid extraperitoneally on January 6, 1948. The bacteriological report was as folows: *Swab from bowel at operation*: Light growth of achromobacter.

He did very well postoperatively and was discharged from the hospital February 13, with an entirely new outlook on life.

SUMMARY AND CONCLUSIONS

1. Three cases representing four operations, in which uretero-intestinal transplants were performed are presented. Each case was prepared preoperatively with sulfathalidine and streptomycin orally.

2. Cultures taken from the bowel when it was opened at operation showed no growth in one case, and very light growth of the usual flora on three occasions in the remaining two cases.

3. These findings are considered to be of considerable clinical value, as by preoperative preparation of the bowel with sulfathalidine and streptomycin orally, the bacteriological flora therein can be reduced to a non-pathogenic level at the time of operation. Contamination of the peritoneal cavity if it should occur, is then of negligible significance.

4. Case 1 indicates a depletion of alkali and potassium by the intense diarrhœa following the uretero-intestinal transplant. Loss of potassium in an adult is an exceedingly rare complication. This case is also interesting in that an interstitial carcinoma was found in a patient with bladder symptoms in whom a nephrectomy had been done 33 years previously for tuberculosis .

5. Case 2 demonstrates a further complication of ureteral intestinal transplants. In this case the catheter splinting one ureter was lost, and retracted within the ureter. The size of the ureteral orifices within the sigmoid at the time of autopsy were surprisingly small. Dilatation of the ureter had occurred above.

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Royal Victoria Hospital.

The life of a general practitioner is one of attention to innumerable details. In addition to the examination and treatment of his patients, and signing countless certificates, he must remember to write to a specialist about one patient, to engage a nurse for a second, to write to a school about a third, to arrange nursing-home accommodation for a fourth, to order a special drug or instrument for a fifth. On top of this comes emergency and night work, and occasional attendance at police, coroner's or county court. None of these details must he forget. He has to suffer fools gladly, and humour the whims of the fractious. He knows that the words he speaks are repeated by one member of a family to all the friends and relatives, and that his actions and competence are the favourite theme of the market-place and tea-parties. His work is concerned mainly with the common complaints—with colds, fevers, general ill-health in children and the chronic illnesses of old age, the accomplishment of which requires little academic knowledge, but much tact, common sense and knowledge of human nature. . . . *Medical Education*, Ffrangcon Roberts, M.D.

PAIN SYNDROMES OF THE CHEST MUSCLES: RESEMBLANCE TO EFFORT ANGINA AND MYOCARDIAL INFARCTION, AND RELIEF BY LOCAL BLOCK*

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FOR a number of years we have been interested in the rôle played by the voluntary muscles in pain syndromes of the chest and arm.[1]

The first aspect of this problem deals with the relation of the somatic muscles to cardiac pain.[2, 3, 4, 5] Although the existence of viscero-motor reflexes has long been recognized,[6] it is not generally appreciated that closure of the coronary arteries results in reflex spasm of the chest and arm muscles just as acute appendicitis, or any form of the acute surgical abdomen, results in reflex rigidity of the abdominal musculature. Furthermore, it has recently become clear that such abnormal impulses from the heart may lead to the development of a self-perpetuating reflex, known as a vicious cycle, which may be maintained for long periods of time by the skeletal muscles.[5] Thus, in effect, a secondary pain syndrome based on spasm of the somatic musculature may be created by impairment of the coronary circulation. The initiating event of cardiac ischæmia and its sequel of intractable chest and arm pain should not be regarded as independent diseases, even though the connection between them may become so tenuous that at times it cannot be traced. In cardiac pain, it is apparent that it is not enough to think only of the heart, but in every instance, the possible complication of a persistent somatic component should be taken into account.

The second aspect of the problem relates to the patient with chest and arm pain in whom no evidence of organic heart disease can be found, but in whom the diagnosis remains unsettled because the purely somatic pain syndromes which result from fatigue and strain of the voluntary muscles are frequently overlooked. Under these circumstances the patient's activity is apt to be markedly limited and his entire mode of living altered because apprehension regarding the heart is not allayed by the making of a positive diagnosis. Indeed, among the factors which may contribute toward the perpetuation of the somatic pain cycle are probably the prohibition of exercise and the lack of normal use of the voluntary muscles.

Although reports from different parts of the world have called attention to somatic pain syndromes of the chest,[7 to 23] our experience indicates that these disorders frequently have not entered into the consideration of the referring physician as a cause of chest pain. Furthermore, even if it is appreciated that the source of pain may lie in the voluntary muscles, the customary methods of treatment are often relatively unsatisfactory. It seemed to us worthwhile, therefore, to re-emphasize the importance of skeletal muscle disorders as a cause of chest pain, and to call attention to an effective method of therapy, namely, local block of somatic trigger areas by means of procaine infiltration or ethyl chloride spray.

Three illustrative case reports have been selected from a larger group of such cases and are presented in detail.

CASE 1

J.T., a 28-year old, white male, first complained of chest pain in August, 1945, shortly after his discharge from the navy. Intense pain was felt intermittently in the precordial region and was associated with a sense of constriction of the chest. Pain was sometimes related to effort, but often came and went in an apparently freakish manner. Occasionally the patient was awakened at night by a sharp stabbing pain in the precordium, which was relieved by deep breathing. He was seen by several physicians, and after thorough cardiologic examination was told that there was no evidence of heart disease. It was suggested that the pain was psychogenic in origin and was precipitated by the readjustment attendant on his return to civilian life.

In November, 1945, about three months after the onset of precordial pain, a dull ache "like a toothache" appeared in the left shoulder and radiated to the inner aspect of the upper arm and elbow. When we first saw the patient at this time, questioning revealed that the onset of shoulder and arm pain was coincidental with his taking a job which involved writing for long periods on a high shelf. The patient was right-handed, but the papers were held in place with the left hand which was moved infrequently. In spite of the previous negative examination, the patient was still extremely apprehensive about his heart.

Examination at this time revealed a well-developed, but high-strung, tense individual. The heart showed only a soft systolic murmur at the apex. The blood pressure was normal. There was no demonstrable limitation of motion of the neck or of the painful upper extremity. The scaleni muscles and the muscles of the chest and shoulder girdle were in general not tender, but in the left pectoral area several discrete spots of deep tenderness were found. Pressure on these spots set off a dull ache perceived throughout the left chest

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anteriorly and along the medial aspect of the upper arm. There was also a circumscribed, exquisitely tender area in the left infraspinatus muscle, pressure on which induced pain perceived in the shoulder and anterior deltoid regions.

These trigger areas were infiltrated with a 0.5% solution of procaine hydrochloride (total amount 80 mgm.) in physiologic saline. The distribution of referred pain induced by insertion of the needle into the trigger area in each instance corresponded with the spread of pain induced by pressure. The chief sites of infiltration and their respective pain reference zones are shown in Fig. 1. This treatment afforded marked, but not 100% relief of pain.

In January, 1946, the patient accepted another job which involved lifting heavy boxes on to a shelf in a warehouse. Within two or three weeks, he began again to have sharp pain in the left pectoral region. This time, the pain appeared almost immediately after lifting these boxes. Sometimes the pain subsided spontaneously within a few minutes, and sometimes it persisted as a dull ache for hours. There was no nocturnal pain. The patient was not able to come to New York until May, 1946, about 3 months after the recurrence of severe pain. At this time an exercise tolerance test (Master two-step technique) was negative. Results of routine laboratory examinations were also normal. On this occasion the patient was able to delineate two separate zones of spontaneous pain (Fig. 2), which

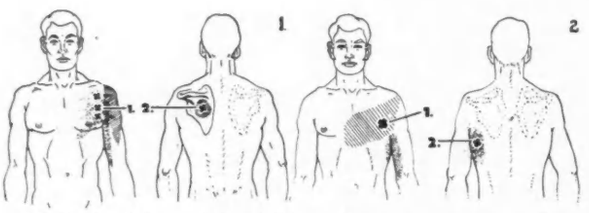

Fig. 1. (Case 1).—Pain reference patterns from trigger areas (x) in (1) pectoralis major and minor muscles (stippled), and (2) infraspinatus muscle (cross-hatched), as noted at first treatment (November 22, 1945). **Fig. 2.** (Case 1).—Pain reference patterns from trigger areas (x) in (1) pectoralis minor muscle (cross hatched), and (2) latissimus dorsi muscle (stippled), as noted at second treatment (May 14, 1946).

appeared independently of each other. One paralleled the lower border of the left pectoralis major muscle and extended from the front of the left shoulder to the lower part of the sternum and slightly across the sternum to the right side of the chest; the other included the lower part of the axilla and the underarm as far as the elbow. Infiltration of an exquisitely tender trigger area, probably located in the pectoralis minor muscle close to its origin from the fourth rib, set off an intense spread of pain, the distribution of which matched closely the spontaneous area of chest pain previously described by the patient. Infiltration of another trigger area located low in the latissimus dorsi muscle induced a reference of pain which reproduced almost exactly the second zone of spontaneous pain in the axilla and underarm. At this time, infiltration of the slightly tender infraspinatus muscle did not set off a clear reference of pain, as it had previously at the first treatment. This finding was in harmony with the patient's statement that shoulder pain no longer troubled him. As on the first occasion, a 0.5% solution of procaine hydrochloride in physiologic saline (total amount 120 mgm.) was used for infiltration. This treatment resulted at once in complete relief of pain.

Follow-up.—There had been no recurrence of pain when the patient was last seen on November 28, 1947. During this period of about 2 years the patient worked continuously as a bookkeeper.

Comment.—This case presents a clear picture of somatic muscle pain which superficially resembled angina of effort. However, the young age and normal cardiovascular findings militated against a diagnosis of pain due to coronary artery insufficiency. The diagnosis was obscure at first because the onset of pain was gradual and lacked a clear relation to voluntary muscle effort. But as the syndrome progressed, it became evident that pain was induced by work of certain skeletal muscles. The relation of pain to body movement in the pectoralis minor syndrome has been emphasized.[21]

CASE 2

M.B., a 68-year old, white woman, was first seen on March 4, 1947, because of substernal oppression with radiation to the neck on effort. The syndrome was of about one year's duration. The pain appeared on walking if she was tired, or on standing for any length of time. On the other hand, when she "felt well", she could sometimes walk long distances, even miles, without pain. It also often occurred on exposure to cold air and on excitement. Following effort, the pain disappeared promptly on resting. There was no nocturnal pain. She complained of frequent palpitation. For several years previously she had had minor episodes of substernal discomfort, but the severity and frequency of these attacks had increased suddenly about a year previously. Shortly after this exacerbation of pain, she was examined by a "heart specialist" who told her that her electrocardiogram was abnormal and that she must expect to have pain for the rest of her life. There never had been any dyspnœa or signs of congestive heart failure.

Pain had also been present in both hip regions for several years. She had taken a series of "baths" at a spa four years previously on account of this complaint. In the last year, pain in the left hip, like that in the chest, had become much worse, and abduction of the left thigh had become quite limited so that she had difficulty in pulling a stocking over the left foot in the usual manner.

There was a history of malnutrition during the war years and until the summer of 1946, while the patient was living in Germany; she had lost about 30 pounds in weight. It was during this time that the substernal pain became a problem. Although the weight loss had been completely regained in the past year and the blood count was normal, the pain had not lessened.

General physical examination was negative except for absence of vibration sense in the toes and reduced ankle jerks. The heart was not enlarged. The blood pressure at the first visit was 120/90; readings at subsequent visits were 115/80 and 100/78. Special examination of the skeletal muscles revealed marked tenderness over the upper part of the sternum and in the right and left parasternal regions for the upper two-thirds of the sternum (Fig. 3); tenderness of the sternal heads of the sternomastoid muscles; marked tenderness of the left adductor longus muscle with limited abduction at the left hip joint. Laboratory examinations were negative except for a basal metabolic rate of minus 16% and frequent premature auricular contractions in the electrocardiogram; the configuration and time relations of the electrocardiographic complexes were otherwise normal.

At the first visit on March 4, the sternal and parasternal regions of the chest were lightly sprayed with ethyl chloride (Fig. 3), according to the interrupted technique previously described.[5, 24] Immediately after spraying, tenderness to pressure in this area had disappeared. After this treatment there was no further

chest pain during the subsequent 3 months in which she remained in close touch with us. At the next visit 3 days later, the chest was again sprayed with ethyl chloride although there was no complaint of pain. At the third visit, 4 days later, the patient described an intermittent "choking sensation", or constriction of the throat, similar to that which used to accompany the episodes of substernal pain. The sternal attachments of the sternomastoid muscles were found to be very tender to pressure, and the front and sides of the neck were sprayed with ethyl chloride at this time. Following this treatment the disagreeable sensation in the throat did not recur. On this date a report of the basal metabolism test was received and appropriate doses of thyroid were prescribed, together with vitamin supplements (B complex, tocopherols and ascorbic acid).

Follow-up.—About 8 months later, on January 23, 1948, the patient returned for examination and stated that there had been no recurrence of chest pain. The electrocardiogram was unchanged; frequent auricular premature contractions were evident. She had soon "run out" of the vitamins, and had taken no medication.

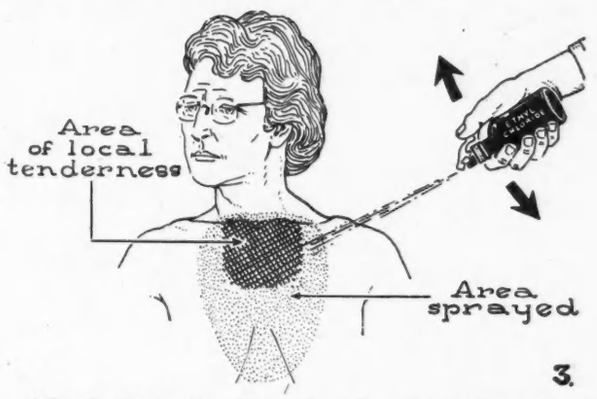

Fig. 3. (Case 2).—Sternal and parasternal areas of local tenderness are cross-hatched, and region of chest sprayed with ethyl chloride is stippled. Note acute angle of stream with skin, sweeping motion of hand and distance bottle is held from chest (12 to 18 inches).

Comment.—This case differs from the previous one in that the pain syndrome resembled true effort angina rather than "pseudo-angina"; pain was substernal and was brought on by walking, cold and excitement and never by movement of any particular part of the body. Complete relief in this case was secured by ethyl chloride spray rather than by local procaine infiltration.

As we have indicated,[5] a striking response to local block therapy can not be used as a basis for deciding whether pain is of visceral or somatic etiology, and often a definite conclusion regarding its source cannot be reached. We believe, however, that in this patient the evidence points toward a somatic, rather than a cardiac origin of chest pain. In the first place, evidence of coronary artery disease was lacking in the electrocardiogram, since the only abnormality was the presence of auricular premature contractions; secondly, pain elsewhere in the body became worse at the same time that chest pain became troublesome; this association of muscular spasm in the lower extremity suggested that some general factor might underlie both pain syndromes; thirdly, the restriction of activity due to substernal pain varied tremendously at different times; when the patient was tired, walking only a few blocks induced pain, but when rested she could walk for miles without pain. In our experience this enormous variation in disability over short periods of time, even from day to day, is characteristic of painful skeletal muscle disorders. Although such variability is also noted in a certain number of cases diagnosed as angina pectoris, it is quite possible that in this latter group there exists a large somatic component of the cardiac pain.

One of the well-known hazards of administering thyroid hormone to patients with coronary sclerosis is the danger of inducing or aggravating angina pectoris.[25] Therefore, in this case which clinically resembled effort angina, one might well question the wisdom of giving thyroid with the object of raising the basal metabolic rate above its level of minus 16%. On the other hand, our experience with a large group of subjects with chronic or recurrent muscular pain in different parts of the body indicates that a large proportion of them have low basal metabolic rates, and that under these circumstances it is extremely difficult to free them from pain by any form of therapy unless sufficient thyroid is given to bring the metabolic rate up to normal (above minus 5%).

CASE 3

H.H., a 71-year old, white male, was hospitalized on October 24, 1941, because of pain in the precordium and shoulder of 4 weeks' duration. He had been well until 4 weeks prior to admission when, while walking in the park, he suddenly experienced a severe pain in the left chest with intense radiation to the left shoulder and arm as far as the elbow. The onset was accompanied by cough and general weakness. He sat down for a while on a park bench and soon felt somewhat better. However, the precordial and arm pain continued without remission. It was aggravated by deep breathing and by almost any movement of the body. The patient said that he favoured "the left arm" and preferred to hold it tight against the chest.

On admission, physical examination revealed a thin elderly man who appeared sick, anxious and in pain. There was no dyspnœa, orthopnœa, or cyanosis. The lungs were normal. The heart was not enlarged. There were no thrills or murmurs. The pulse and ventricular rates were 84 per minute, and the rhythm was regular. The blood pressure was 100/84. There was no enlargement of the liver or spleen, and no œdema. According to subsequent questioning, limited motion was present at the left shoulder joint, but was not noted in the record. The tentative diagnosis on admission to the hospital was acute myocardial infarction. This was based chiefly on the mode of onset, and was not confirmed by the subsequent course or laboratory findings.

There was no fever during hospitalization. The blood sedimentation rate was 10 mm., and 6 mm. at the end of an hour on the first and third hospital days, respectively. The white blood count was 9,400 with 58% neutrophiles. X-ray of the chest was negative. The electrocardiogram was normal. The venous pressure was 10 cm. of water. The circulation times were normal; arm to tongue (calcium gluconate) 17 sec., and arm to lung (ether) 5 sec. X-ray of the dorsal spine showed slight productive changes along the margins of the thoracic and lumbar vertebræ. X-ray of the right shoulder showed a small calcific deposit in the region of the subacromial bursa.

When we first saw the patient on October 31, seven days after admission, he was brought to our treatment room on a stretcher. Re-evaluation of the history led us to question the diagnosis of an acute cardiac disorder because of the unremitting character of the pain and the marked degree of limitation of motion at the shoulder. Furthermore, corroborative evidence of a recent myocardial infarction was lacking in the electrocardiogram and other laboratory tests. In addition, there had been an episode of similar, but less severe pain and restricted motion in the right shoulder, which began ten days after the onset on the left side and which disappeared in a few days.

At this time, motion at the left shoulder joint (Fig. 4) was almost totally limited. Abduction was restricted to 26 degrees. The range on extension was practically zero; by bending the elbow, the hand could be brought as high as the face only. Internal and external rotation at the shoulder were likewise completely limited, and attempts at making these movements were excruciatingly painful. The range of extension and flexion of the elbow, and of supination and pronation were normal, but all motions of the arm and also of the neck and trunk were extremely guarded.

Fig. 4. (Case 3).—Photographs showing maximum range of motion at left shoulder joint in relation to treatment by local block of trigger areas in left posterior scapular and interscapular muscles. Note marked limitation just before treatment and normal motion shortly afterward. Photographs a and b were taken before infiltration, and c was taken 20 minutes after infiltration.

Palpation of the muscles of the left shoulder girdle disclosed a trigger area in the left infraspinatus muscle, pressure on which induced an intense accentuation of the spontaneous pain in the shoulder and arm (Fig. 5). Another trigger area from which a wide reference of pain could be induced by pressure was found at a point just medial to the scapula at the level of the spine of the scapula. This trigger area was judged to be located in the posterior superior serratus muscle because of its depth as demonstrated by local injection (Fig. 5). There was also generalized, low-grade tenderness to pressure of all of the muscles of the shoulder girdle, and also of the cervical and upper thoracic spinal muscles on the left side. There was a point of exquisite tenderness directly over the spinous process of the seventh cervical vertebra. There were no abnormal neurologic findings.

A total of 5 c.c. of a 1%* aqueous solution of procaine hydrochloride was used for infiltration of these two outstanding trigger areas. The injections were accompanied by an intense spread of pain to the chest, shoulder, arm and even to the neck. This was followed at once by a feeling of profound weakness and heaviness of the arm; for a few moments the patient could raise the arm with a quick movement, but a sustained effort could not be maintained. At this time it was noted that the widespread generalized tenderness of the muscles had entirely disappeared, as well as the tenderness over the spinous process of the seventh cervical vertebra.

Within 20 minutes after the start of the treatment, another photograph was taken (Fig. 4), which shows that the range of motion had been restored to normal. The patient could swing his arm rapidly in a full circle without pain. He helped wheel his stretcher back to his room and greeted the resident physician by lifting him up in his arms and carrying him around the room. There was no further treatment, and 3 days later (November 3), the patient was discharged to his home free from pain.

Follow-up.—The patient has now been observed for 6 years. There has been no recurrence of pain in the chest or upper extremity. On March 12, 1947, at the age of 72 years, the patient was last re-examined. The only change was that a low T_1 had appeared in the electrocardiogram.

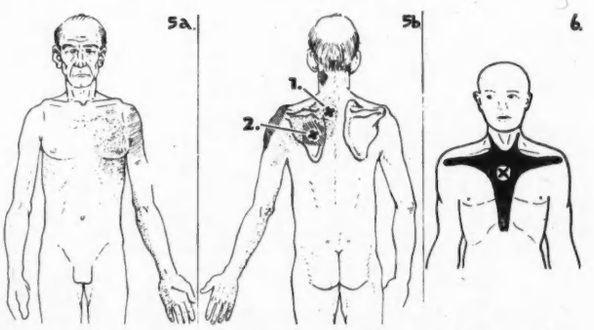

Fig. 5. (Case 3).—Pain reference patterns from trigger areas (x) in (1) posterior superior serratus muscle (stippled), and (2) infraspinatus muscle (cross-hatched), as noted at treatment on October 31, 1941. **Fig. 6.**—Composite pain reference pattern of sternalis muscle from trigger areas located at (x).

Comment.—In this case the resemblance of the clinical picture at the onset to acute coronary thrombosis is striking. This diagnosis was suggested by the fact that the patient was in the coronary disease age group, by the suddenness of onset of intense precordial pain with ulnar radiation, and by the lack of any relation to special use or obvious strain of the voluntary muscles. It is obvious that when a patient who is 65 years of age presents himself with such a history, the burden of proof is on the physician to exclude acute myocardial infarction. Nevertheless, this case illustrates the need for consideration of somatic causes of the symptomatology even in the presence of a presumptive diagnosis of acute coronary thrombosis. The clue to the etiology, as subsequently demonstrated, lay in the fact that extreme limitation of motion at the shoulder joint was present from the onset. In our experience, the "frozen shoulder syndrome" has occurred as a *late* sequel

* For reasons stated,[26] we now prefer a lower concentration of procaine hydrochloride for infiltration, namely, 0.25 to 0.5%.

to myocardial infarction and not coincidental with the development of this cardiac lesion. This lag in the appearance of post-infarction shoulder pain has also been noted in a current review.[27]

DISCUSSION

Our recent studies[4, 5] focus attention on the fact that pain clearly of cardiac origin can be abolished, either temporarily or permanently, by measures which block the somatic trigger areas concerned in the reference of pain from the heart. The present report deals with the differential diagnosis and treatment of pain syndromes of the chest which are primarily *somatic* in origin, but which resemble effort angina or acute myocardial infarction.

In the first place, our observations are in conflict with the frequently encountered view[8, 11, 18, 20] that when pain on effort (walking) is *substernal,* it is necessarily due to coronary artery disease, whereas when it is located in the *precordium,* it may originate from either cardiac or somatic causes. In the second place, we have found that certain other criteria which are often accepted as pointing toward a cardiac etiology of chest pain, can no longer be regarded as reliable. The intensity and quality, as well as the distribution, of so-called cardiac pain can be reproduced in every detail by the referred pain momentarily induced by stimulation of trigger areas in the chest muscles.[4, 5, 6, 16] Furthermore, we can no longer depend with certainty on transient electrocardiographic changes if the electrocardiogram was taken during an attack of pain, as it often is, since extracardiac pain, induced for example by work-ischæmia of the forearm muscles, may temporarily produce abnormalities of the T-waves compatible with a diagnosis of coronary insufficiency.[28] Even relief by nitrites does not necessarily signify that one is dealing with coronary artery pain, because a placebo sometimes appears to be equally effective in effort angina,[29] and because nitrites have been found occasionally to relieve skeletal muscle pain.[9, 11]

It is sometimes implied[21] that the absence of pain on special movement of the trunk or arms excludes a somatic etiology of chest pain, but this conclusion is not valid since trigger areas which include the chest in their pain reference patterns, may develop in certain locations without producing any demonstrable restriction of motion. The rudimentary sternalis muscle on the face of the sternum is an illustration of this fact; the composite reference pattern of this muscle as observed in a group of patients is shown in Fig. 6. It should be emphasized that conversely, the presence of limitation of motion does not rule out the diagnosis of a post-cardiac pain syndrome.

What diagnostic criteria, then, remain to distinguish somatic from cardiac pain syndromes of the chest? In acute episodes of pain suggestive of myocardial infarction, the presence or absence of signs of tissue necrosis and circulatory collapse are of the utmost importance in weighting the diagnosis in one direction or the other. The absence of trigger areas would seemingly rule out the skeletal muscles as a cause of pain, unless of course, the trigger areas happened to be located in the retrosternal striated muscles of the chest wall where they would not be accessible to palpation. That the pain syndrome is primarily somatic in origin is often suggested by a "rheumatic tendency" of the patient, or the finding of painful motion elsewhere in the body attributable to trigger areas in appropriate muscles. In chronic effort syndromes, the most valuable positive sign of a somatic disorder is an enormous variability in the exercise limit, or capacity for effort without pain, over short periods of time, as noted in Case 2. In our experience, such variability is rarely seen in coronary artery disease; in true effort angina, just as in intermittent claudication, pain appears after walking approximately the same distance, within narrow limits, from day to day. On the other hand, if the distance which the patient can walk fluctuates widely, it is likely that there is a large somatic component in the etiology of pain.

A pertinent therapeutic problem is the question whether pain in cardiac disease is a protective mechanism, and its removal therefore contraindicated. We have been led to conclude[5] that pain itself may reflexly diminish the calibre of the coronary arteries and so contribute to further damage of the myocardium.[3, 30] In addition, when pain is not suppressed, the excessive restriction of activity which may result, often leads to physical and mental deterioration of the patient. In our opinion local block therapy[24] is indicated for the relief of chest pain whether the somatic manifestations are believed to be skeletal or visceral in origin, and even when there is so much evi-

dence in support of both etiologies that the primary cause of pain cannot be positively established.

SUMMARY

1. Three case reports demonstrate the striking resemblance which pain syndromes of the chest muscles may bear to effort angina and acute myocardial infarction.

2. Intractable chest pain which originates in the skeletal muscles may be relieved by local block therapy (procaine infiltration or ethyl chloride spray) in a spectacular manner.

3. Relief of pain by these measures does not prove that the cause of pain is primarily somatic, since under suitable conditions cardiac pain may likewise respond to local block of the appropriate somatic structures.

4. Other criteria which differentiate somatic from cardiac etiologies of chest pain are discussed.

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The detection of early malignant lesions of the breast if often impossible for the layman and even difficult for the physician because of the small size of the growth. Three cases are presented to illustrate the need of a careful examination of the axilla of all patients despite negative breast findings. If axillary nodes are found they should be removed for pathologic examination, and if found to be malignant the advisability of a radical breast amputation should be given most serious consideration. Carcinoma of the Breast in the Absence of Clinical Breast Findings. Jackson, A. S., *Ann. Surg.*, **127**: 177, 1948.

THE CARE OF PARAPLEGIC PATIENTS IN GENERAL HOSPITALS

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MUCH of the recent literature on paraplegia deals with the management of the paraplegic patient in specialized centres. However, many patients are not fortunate enough to be admitted to such centres until their convalescence has progressed through the important preliminary stages. Almost all paraplegics receive their first treatment in general hospitals, where the staff has had no opportunity to carry out paraplegic care. The advanced work in paraplegia in Canada is being done at such special centres as Lyndhurst Lodge, Sunnybrook, and Deer Lodge. These units resulted from the combined efforts of D.V.A. and the Canadian Paraplegic Association. Until mid 1947 they dealt mainly with paraplegia incurred during service in the armed forces. As the patients of this type gradually completed their retraining program, more beds became available for civilian use.

Statistics show that, although the initial expenditures of active treatment necessary to restore these patients to self-sufficiency in active society are high, this policy is, over a long term, cheaper than maintaining the patients and their families as public wards. This article arises from the problems encountered during the treatment of six paraplegic patients at the Ottawa Civic Hospital. No conclusions may be drawn from this small series of cases, but the lessons learned, backed by the recorded experience of others, are worthy of attention.

The fundamental of paraplegic care—*teamwork*—should be readily available everywhere. Without teamwork, the most expensive equipment and the best of intentions are ineffectual. This fact should be clearly understood by the patient, nurses, orderlies, dietitians, social service workers, physiotherapists, interns, the urologist, orthopædist, and general surgeon. A general hospital team's objective is to prepare the patient for transfer to a paraplegic centre, at the earliest possible date, without the development of urinary sepsis, decubitus ulcers or contractures. The team has failed if any of these complications develop. It is for the specialized centre to decide upon the necessity of pro-

cedures such as rhizotomy, and the type of final mobility the patient will attain. Any member of the team who feels that he alone is competent to supervise the patient to complete recovery is merely salving his ego, at the expense of the patient's future.

For the majority of the patients coming to a general hospital, the origin of the paraplegia is traumatic. Shock, soft tissue damage, and fractures should be treated in the usual manner. Paraplegia should be suspected if there is flaccid paralysis with abolition of tendon jerks and plantar responses. The patient may complain of a zone of hyperæsthesia, or of tight band-like sensation just above the lesion level.[1]

Care of the genito-urinary tract.—If the patient suspected of paraplegia has not voided within eight hours after admission, and has a percussable suprapubic mass, a Foley catheter, never larger than 18 French, should be aseptically inserted into the bladder. A larger catheter will not prevent leakage. It will predispose toward sphincter atony and pressure sores of the urethra, which is even less resistant to pressure damage than the tissues over the sacrum and trochanters.

The greatest single advance in paraplegic care is automatic bladder irrigation, first described by Laver[2] in 1917, and improved by Munro and Hahn.[3] The literature[4, 5, 6] contains descriptions of many variations and improvements of the original model. At the Ottawa Civic Hospital we used a simple, cheap, easily assembled irrigator. Lightweight brown intravenous type rubber tubing, which does not kink easily, is used. The irrigator jar and connecting tubing are autoclaved in packs prior to use. When in use, this equipment should be dismantled, cleaned, and re-autoclaved every seven days. Tidal irrigation is commenced immediately, with the siphon level set at 1 to 2 cm. above the level of the pubis. When necessary, the tidal irrigator may be temporarily disconnected to position the patient for x-rays and operative or manipulative procedures.

Cystometry.—As soon as possible, a cystometrogram (following the Munro routine outlined below) must be done to estimate the functional capacity of the bladder.

The following routine for cystometry is suggested by Munro.[7]

1. Empty the bladder.
2. Clamp tubing leading to siphon.
3. Release the clamp on lead-in tubing to allow measuring solution, either solution G, solution M (citric acid 32.35 gm., sodium carbonate (anhydrous) 8.84 gm., magnesium oxide 3.84 gm., distilled water q.s. ad 1,000 c.c. autoclave before use, tint with methylene blue) of Suby and Albright,[8] or 1.5% boracic solution to flow from a calibrated glass container, 50 cm. above bladder level, at the rate of 90 drops per minute (a faster rate provokes an unphysiological bladder response) through the Murphy drip, tidal irrigator and via the Foley catheter into the bladder.
4. After running in each 25 c.c. of fill, clamp the inlet tube and note manometer level.
5. At each change in intravesical pressure, recorded by the fluid level in the glass manometer, note on paper: (*a*) height of fluid in manometer; (*b*) amount of fluid that has run into the bladder; (*c*) the point at which any leakage occurs about the catheter.
6. Continue until 400 c.c. of fluid has been run into the bladder. If there is cause to believe that there will be reflux up the ureters, due to hypertrophy or fibrosis of bladder wall, or if there is a history of cystitis, a smaller amount of fluid (100 to 200 c.c.) must be used.
7. Disconnect the catheter and allow the bladder to empty. Hold the open catheter end at the level of the pubis.
8. When this flow ceases, lower the end of the catheter and measure any residual that siphons out.

The four sets of figures: (*a*) the amount of fill; (*b*) the manometer fluid level for each 25 c.c. of additional fill; (*c*) the manometer fluid level when any bladder contraction occurs; (*d*) the residual, will give all information necessary for bladder care in a general hospital.

The interpretation of these data is as follows: (1) An emptying contraction occurs when intravesical pressure reaches 50 cm. of water or when there is leakage about the catheter. Classic emptying contractions develop solely in normal or reflex bladders. (2) Minimal capacity of the bladder is the amount of fill that has collected between the first and the second emptying contractions. (3) The curve of basic tonus indicating the irritability of the detrusor muscle is determined over a period of weeks by subtracting the lowest intravesical pressure from the highest, noting the amount of fill and plotting a graph of the two columns of figures on co-ordinate paper.

Reflex anal contraction resulting from pricking either the glans penis or perineal region denotes a normal anal or bulbocavernosus reflex. This reflex is present only in the absence of spinal shock and when the internal pudic nerve (or pudendal - S-2-3-4) and its spinal cord connections are intact. Presence of this reflex means the external urethral sphincter is under either voluntary control or involuntary reflex control. Absence of the reflex anal contraction, if spinal shock is not present, denotes paresis of the external urethral sphincter.

Cystometry, which includes measurement of residual, must be done at least every three weeks

to determine the stage present, and to follow the patient through each step toward optimum functional activity. Increasing bladder tone, in the absence of infection, should be accompanied by gradually increasing the siphon level. If bladder capacity diminishes, and/or residual increases, the cause must be located and corrected. Urinary tract calculi, cystitis and suprapubic drainage reduce bladder capacity. Hypertrophy or fibrosis of bladder muscle, or pseudomedian bar formation of the internal urethral sphincter cause urinary retention. These conditions must be detected quickly and treated before stasis leads to calculus formation.

A patient confined to bed tends to mobilize calcium, thus increasing urinary calcium content. This, in the presence of genito-urinary tract sepsis, is believed to be a potent factor in stone formation. A paraplegic patient with a temperature elevation of 99 to 100° complaining of generalized, dull, oppressive, abdominal discomfort, which is increased by palpation or percussion of either costo-vertebral angles, should have an immediate KUB x-ray film. Each paraplegic patient should have a KUB film every three months, and an intravenous pyelogram every six months, even if the above indications are absent. Cystoscopy and retrograde pyelography should be done if indicated. This routine detects early asymptomatic calculus formation.

Decubitus ulcers.—The development of decubitus ulcers is an urgent indication for critical re-assessment of the entire routine of paraplegic care. Action must be immediate and aggressive.

In 1936 Lewis[9] demonstrated that ischæmia of six to twelve hours damages the skin, while continuous ischæmia for twenty-four to forty-eight hours produces necrosis of the skin. In paraplegics who have lost their normal muscular tone and elasticity of the subcutaneous tissue pad, a complete obliteration of the vessels between the skeletal prominences and pressure points occurs. Trauma, soiled bed linen, disuse atrophy of the interposing padding, skin and tissue alterations, due to toxæmia and hypoproteinæmia, are additional factors facilitating embarrassment of local circulation. Each patient's vulnerability to decubitus ulcers is directly proportional to the degree of severity of these conditions. This fact must be explained in understandable terms to the patient, nurses, and orderlies carrying out the daily routine paraplegic care. The importance of a rigidly adhered-to turning schedule must be constantly re-emphasized. Routine back care must be fastidiously performed twice daily, and whenever the patient becomes soiled. Pressure points in recent paraplegics must be relieved every three hours day and night.

Our first case at the Ottawa Hospital was a traumatic dislocation of the body of the eighth thoracic vertebra and compression fracture of the ninth thoracic vertebra. The patient was immediately placed in a hyperextending, bivalved plaster bed and placed on a q.3h. turning routine. This was not rigidly adhered to, and decubitus ulcers over both patellæ developed. Attempts to treat them with moist penicillin compresses failed. The tissue became macerated, and the areas involved extended. Finally, the sloughing areas were controlled by dry heat, and then covered by a sliding flap graft.

Most workers[10] agree that ointments and wet dressings macerate the skin. If a decubitus ulcer develops, use dry heat. When there is clinical evidence of clean margins, absence of local infection, a clear urinary tract and normal temperature, cover the defect following excision of the edges with a split thickness skin graft or a rotation flap.[11] Spontaneously healed ulcers leave easily traumatized scars. Do not apply wet dressings or ointments to paraplegic bedsores, and do not wait for them to heal spontaneously.

Diet.—Routine hospital diet is inadequate for the requirements of most paraplegic patients.[12] The order "high protein, high carbohydrate, high vitamin diet" does not remedy this lack. From the first hospital day, one of the team, preferably a dietitian, must be made solely responsible for the diet. At the earliest possible moment, the patient should be weighed. A chart of the weight, determined every seven days for the first two months and then every 14 days, is the simplest and most accurate means of assessing dietary adequacy. Poer's[13] statement that 74% of wartime paraplegic patients arriving in the United States following injury received overseas had developed marked emaciation with an average weight loss of 42.5 pounds per person gives one an indication of the extent to which the nutrition of the paraplegic patient can deteriorate.

The diet must prevent or correct hypoproteinæmia. That is, the diet must never allow negative nitrogen balance to exist. Mecray[14] has demonstrated that hypoproteinæmia prolongs the emptying time of the stomach. Barden[15] demonstrated that the same factor impairs small intestine function. With these two findings in mind, one can realize the difficulty with which the hypoproteinæmic patient ingests the required large amounts of food. Some of the objections to bulk, flavour and cost may be overcome by the use of concentrated food sources of protein. Soybean flour, which can be used in pancake and muffin mixes, soups and pastry products, contains 37 to 45% protein.[16] Peanut flour, which can be blended in hominy grits, rolled oats and corn-meal, has 50 to 60% protein.[17] This flour may function as a binder in meat loaves, sausages, and as a base in soup concentrates. Dried yeast, an excellent vitamin source, is approximately 45% protein. This source may be used with syrup, peanut butter, or in baked products such as bread, cookies and doughnuts.[18] Dried gelatin yielding up to 85% protein may be added to hot beverages and soups. Bauman[19] devised a high protein beverage consisting of milk, frozen egg whites and powdered milk (Dryco). One glass (200 c.c.), supplying 20 grams of protein, provided 240 calories.

As the patient's condition becomes stabilized, excessive weight gain may easily occur, but this must be avoided. This development creates an unnecessary handicap in the performance of basic exercises, wheel chair manœuvres, crutch-walking and brace ambulation. An ideal objective is the maintenance of the patient's weight within ten pounds of his computed basic weight.

Care of the bowels.—Many facts remain to be learned concerning the altered bowel function in paraplegic patients. Best and Taylor[20] state that section of the spinal cord above the sacral segments causes relaxation of the colon. This is felt to indicate that constant tone augmentor impulses are received from higher centres. If the pelvic nerves are severed some time after spinal transection, colonic relaxation results. This is believed due to sacral segments acquiring control of colonic tone during the interval following cord transection. This theory suitably accounts for the gradual improvement in colonic function, usually noted as the patient progresses towards a more active life. Derangement of bowel function is less marked if the cord lesion is not complete. Generalized increase of muscular tone is an important additional factor in the restoration of regular bowel evacuation.

While in a general hospital the patient should receive bland enemata (*i.e.*, milk and molasses, saline or hydrogen peroxide) at a routine time every second day. Prolonged use of more irritating agents will eventually cause a troublesome chronic proctitis. At the Ottawa Civic Hospital, we found pituitrin or prostigmine helpful if given intramuscularly fifteen to thirty minutes preceding enemata administration. Teaching of self-administration of enemata should be done at the specialized paraplegic centre later in the patient's convalescence. Laxatives such as gelucil, petrolagar, agarol, liuid paraffin, or milk of magnesia may be used. The patient, after a trial of each type of agent, should be allowed to adopt the most suitable to his individual use. If regular bowel movements are obtained by this means, enema administration may be discontinued. Fæcal impaction in paraplegic patients is the commonest cause of frequent loose bowel movements.

Physiotherapy.—The aim of a physiotherapist in a general hospital is to prevent contractures and muscular deterioration, particularly of the arm and upper trunk musculature. It is the physiotherapist's responsibility to see that a foot board is constantly in place to prevent the pressure of bedding causing foot contractures. As soon as the patient's general condition permits, simple reconditioning exercises should be done under supervision twice daily. Gradually the schedule should be expanded to include rolling from side to side, resistance pulls against weights on pulleys, and trunk lifting by means of overhead bed bars.[21] The active physiotherapy department at the Ottawa Civic Hospital organized wheel chair and mat classes. However, if the general hospital patient is able to engage in these activities, he is ready for transfer to the paraplegic centre.

Psychological aspect.—The wide variety of mode of onset of paraplegia, the patients' personality factors, their background and their subsequent adjustment allow only generalized statements concerning the psychological aspect of management. The variables must be considered individually in each case. A practical, sympathetic, continued understanding of each

patient's problems will go furthest in aiding his re-adjustment. Psychoanalysis and psychiatric consultations will do little to alter the basic pattern of the adult patient.

The necessity of dependence in some degree upon others is soon apparent to the paraplegic patient. Obviously overindulgent pity and attention quickly evokes an unpleasant reaction in most patients. This attitude is not to the pity, but to the consciously or subconsciously perceived implication of the unfortunate and dependent nature of this patient's predicament.[22] Neglect and disregard, on the part of relatives, nurses, orderlies, interns and staff doctors, cause even more intense bitterness.

In most general hospitals, nurses and often inept orderlies are largely responsible for the care of paraplegic patients. In their normal daily routine, the nurses, unless specially detailed, are frequently unable to devote the time required for the individual paraplegic care, without neglecting other patients. At the Ottawa Civic Hospital, we had one patient who, when turned, required the assistance of two (and frequently three) nurses and an orderly or intern. If this personnel was unavailable, she was not turned q.3h. The appointment of a regular team and attaching a time schedule to the patient's bed overcame this difficulty.

To the patient who but a short time previously had been free and active, claustrophobia is not an idle or technical word as he lies confined to bed. Frequently, daily visitors stand grouped around the patient's bed, peering down. If relatives or doctors are visiting paraplegic patients, they should sit down at a convenient distance from the bed, at a level equal with or lower than the patient, in a position where the patient can easily see them. This assists the patient in maintaining a freer, more natural attitude.

Everyone's daily experiences of varied degrees of frustration are intensified in the paraplegic patient. Having to wait for the nurse or orderly to bring a bed-pan, or to assist him on to the toilet, finding the meal cold or unpalatable, being refused something he formerly could get himself, inaccessibility of facilities to see a motion picture, inability to engage in a former sport or hobby, all can arouse impotent anger and deep despair. The patient may interpret any of the above, or similar situations, as indifference or neglect. His reaction may be one of temper tantrums, sulking, refusing to take medicine, or offering multiple excuses for not engaging in his daily routine. This adds to any difficulty in maintaining the extremely necessary high degree of motivation. The difficulties are more acute in a general hospital, where the paraplegic patient is in close association with other patients who are making relatively rapid progress toward complete recovery and discharge. At a paraplegic centre, where all general hospital paraplegic patients should eventually be sent, the patient is in close daily association with others similarly afflicted who are combating the same problems and situations and reaching an adequate solution.

In male patients, the loss of sexual function (ejaculation) should be discussed and explained. At the present time, clinical research being done in England and the United States may change our present concepts of this depressing situation.

"Into the fertile field of the indefinite future, these patients project their hopes of eventual complete recovery".[22] Caution is imperative in discussing prognosis. Over-optimism or over-pessimism on the part of attendants can swing the patient from the heights of elation to the depths of despair. In a traumatic case, where the cord is definitely known by surgical exploration to be irreparably damaged, the prognosis can be given and should be discussed with the patient. A positive statement, unless grounded on fact, is dangerous.

More difficult problems are those cases of disseminated sclerosis passing through periods of exacerbations and remissions in their slow downhill course. In all cases, as long as improvement of function and physical ability improves, the repeatedly reassured patient must adhere to a definite pre-planned daily routine of exercise, physiotherapy and self care.

Restoration to society.—Except when insurmountable major physical defects exist, the treatment of the patient has failed, no matter what degree of physical fitness and accomplishment is attained, unless he is discharged to resume a useful, active part in community life. During convalescence, the patient must be given a definite objective for which to plan actively and constructively. Otherwise, eventual physical and mental deterioration is inevitable. In all cases, the entire mode of living will be altered radically. Mindful of these facts, a responsible member of the team should seek to interest such organizations in the patient's own community as the Kiwanis, Rotary, Knights of Columbus, and

similar groups, in each patient's problems connected with return to community life. It is short-sighted to wait until the patient has reached the zenith of his recovery before asking such organizations to be interested in assisting the patient's social re-establishment. The club members' interest and activity will be greater if a delegated club member follows the patient's progress from a state of relative helplessness to his return to active society. These clubs will more eagerly survey job possibilities, and assist in furnishing such necessary extras as braces and wheel chairs.* The assistance of such organizations speeded the return to community life of several of the group of paraplegic patients treated at the Ottawa Civic Hospital.

SUMMARY

1. A policy for general hospital care of paraplegic patients is outlined.

2. The complexity and multiple details of the problem, and necessity for constant co-operation are stressed.

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* The Everest-Jennings wheel-chair, with or without detachable side-arms, is the most satisfactory type available. It weighs approximately 32 pounds and can be collapsed to a width of 10 inches. Information concerning this equipment may be obtained from the Everest-Jennings Company, Los Angeles, California, or the Canadian Paraplegic Association, Toronto.

Appearance of whooping cough in a locality should be the cause of much more general concern than is usually expressed. For whooping cough is not just an unpleasant experience in a child's life—it is a real menace to health, one of the most serious of the contagious diseases. A child should be immunized against whooping cough at the earliest opportunity. This will provide complete protection for from seven to nine out of every ten, and the remainder will suffer milder and shorter attacks.

THE SURGICAL TREATMENT OF INTRAVENOUS CLOTTING*

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THIS subject is still a very controversial one, and I can only place on record my impressions of the matter. These impressions are, however, a result of the evaluation of the observations of others.

Before anyone can intelligently treat any condition, one must have some reasonably clear-cut idea of what that condition is, and what the factors are which bring it about.

ETIOLOGY OF INTRAVENOUS CLOTTING

Predisposing factors. — (*a*) Cardiovascular disease; (*b*) advancing age; (*c*) foci of infection; (*d*) obesity; (*e*) debility; (*f*) anæmia; (*g*) varicose veins; (*h*) seasonal variations; (*i*) geographic factors and a number of others.

Precipitating factors. — (*a*) Trauma, with vascular changes and circulatory retardation; (*b*) absorption of noxious substances (polypeptides) from injured cells resulting in changes in the blood; (*c*) infection (gross); (*d*) inertia and posture leading to venous stasis.

Cardiovascular disease.—Some authors[1] emphasize the statement that the incidence of postoperative thrombosis is dependent more upon the heart and the state of the general circulation than it is upon the disease process or the operation. Others[2] have found that in 90% of their reported 91 cases of fatal pulmonary embolism there was pre-existing cardiovascular disease at the time of the fatal seizure.

Age.—According to a number of authorities the incidence of thrombosis is greater in the later decades of life. The vast majority of the reported cases are between the ages of 40 and 60 years. It is comparatively rare under 40 years of age.

Seasonal influence. — Some of the older authors[3, 4, 5] believe that the incidence is higher in the spring and fall, and attribute this to the prevalence, at these seasons, of the "grippal" infections.

Geographic factors. — This is emphasized by some authors,[6] who report that the average incidence of thrombosis is 0.75 per 100,000 of population in northern states, whereas it is

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only 0.41 in southern climates. One explanation offered for this phenomenon is a hyperexcitability of the circulatory system as a result of repeated vasospasms due to cold.

Obesity. — All surgeons tend to regard the obese patient as a poor surgical risk, and whether or not this is due to a fatty infiltration of the heart muscle, resulting in poor circulation, or merely to lethargy and inertia, it has been reported by Barker and his coworkers that in a series of 3,680 patients upon whom intestinal operations were done, thromboembolism occurred in 3.2% of those who weighed under 200 pounds, and in 7.1% of those heavier than this.

Debility.—Here, of course, there is a variety of factors grouped under one heading; circulation is poor, there is often disease of the vessels, the patient is lethargic and moves little, and there may also be changes in the blood, leading to an increased likelihood of clotting.

Varicose veins. — Stasis in this condition, naturally favours clotting. However, without labouring these factors further, it will be noted that though a multiplicity of pre-disposing and exciting causes is adduced by an impressive battery of authors, and indeed many more could be cited, all these causes may be essentially narrowed down to two:

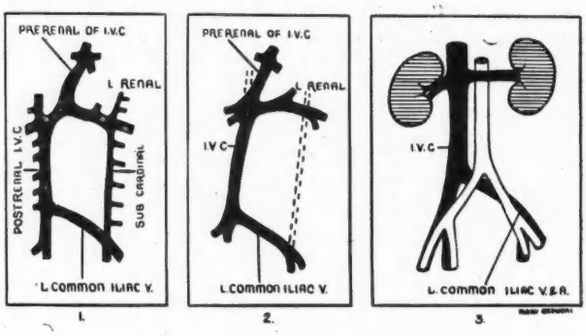
(1) *Deficiency of circulation* due either to cardiac insufficiency, venous stasis or vasospasm. (2) *Changes in the blood*: (*a*) changes in the calcium-potassium ratio; (*b*) changes in the albumin-globulin ratio with increased globulin; (*c*) Increase in both the number and agglutinability of the thrombocytes.

The increase in the number of thrombocytes is a normal postoperative reaction of the body. It has been demonstrated by clinical studies[7] that there is a decrease for approximately the first five days; when they have presumably been "used up" in overcoming the operative interference; and then a massive increase between the eighth to eleventh days.

The increase in agglutinability is thought to be due to changes in the electrical charges in the formed elements and a loss of normal repulsion. This, in turn, is said to be a result of the changed albumin-globulin ratio, and this change itself is probably due to the absorption of polypeptides released by tissue cells damaged by the operative procedure. The more trauma to tissue, the more likelihood of clotting (a plea for *sharp* dissection as opposed to blunt). The serum peptidase may be used as a guide to the amount of aseptic cell destruction which has gone on.

ANATOMICAL CONSIDERATIONS

It is common knowledge that intravenous clotting occurs much more commonly in the left leg. The figures are roughly 65% left—25% right—10% both. The reason is, of course, mainly anatomical. In the two-month embryo there are two caudal veins which fuse, with an anastomotic vein below (Fig. 1). That portion of the left caudal vein between the fusion and the anastomosis later disappears (Fig. 2). Hence the ultimate left common iliac vein is the smaller of the two and enters into the inferior vena cava at a much more acute angle than does the right.



DEVELOPMENT OF LEFT COMMON ILIAC VEIN

In addition, the left vein is crossed by the right common iliac artery, and is also liable to be subjected to pressure by a loaded sigmoid or rectum (Fig. 3).

THE PATHOLOGY OF INTRAVENOUS THROMBOSIS

When one comes to study the pathological processes associated with this phenomenon, one is immediately struck by the fact that there are two separate and distinct processes which may occur. One of these is mainly associated with acute inflammatory changes about and in the vein wall and in this the clotting is of a very secondary nature; the other is that in which there is primarily clotting of blood in the lumen, and with very little involvement of the vein wall.

These two processes have been distinguished by the term thrombo-phlebitis for the former, and phlebo-thrombosis for the latter.

Thrombo-phlebitis.—This occurs as a result of eventual injury to the endothelium of the vessel because of trauma (mechanical or chemi-

cal) or bacterial invasion. The invasion takes place usually by way of the peri-venous lymphatics, and only rarely, as in cases of pyæmia, occurs primarily within the lumen; and is usually unaccompanied by any particular changes in the blood constituents.

In consequence of this, since the clotting is secondary to acute inflammatory changes in the vessel wall, there is the laying-down of a white, tough, thrombus due to the adherence of leucocytes and fibrin, which is relatively firmly adherent to the vessel wall, and hence, unless in the rare event of suppuration, unlikely to embolize.

Because of the acute inflammatory process, however, there is associated arterial and arteriolar spasm, with pallor and œdema as a result. Ochsner and DeBakey have shown that the pain in acute thrombo-phlebitis is not due to the inflammation and irritation of the vein proper in the main, but to ischæmia induced by severe homolateral arteriolar spasm, resulting from reflex vaso-constriction impulses arising in the thrombo-phlebitic segment of vein, and carried by the sympathetic fibres. This arteriolar constriction accounts for the whiteness of the skin (phlegmasia alba dolens) and because of the anoxæmia, produces increased permeability of the arteriolar and capillary beds, with excessive transudation of fluid from the vascular system into the intercellular spaces and the rapid onset of œdema.

Aseptic ligation of a comparable vein does *not* produce this phenomenon.

Phlebo-thrombosis.—In this condition, in contradistinction to thrombo-phlebitis, there is the formation of an intravenous thrombosis, due mainly to venous stasis and changes in the blood constituents, and with little involvement of the vessel wall. Consequently there is here, a reddish, relatively friable clot, mainly composed of erythrocytes and very similar to coagulation *in vitro*. This thrombus is only casually attached to the vein wall, and embolizes readily. The lack of an acute inflammatory process with its associated pain, fever and vaso-spasm, renders recognition of this condition difficult until embolism has occurred.

This differentiation, of which more will be said later, has great therapeutic significance, in that recognition of these respective processes will permit of the institution of appropriate measures of treatment.

All too often these terms thrombo-phlebitis and phlebo-thrombosis are used as if they were completely interchangeable and were merely synonyms for the same ailment. True, they have in common the factor of intravenous thrombosis, but beyond that are as different as the poles, and so also are their treatments.

DIAGNOSIS

Clinically the diagnosis of acute thrombophlebitis offers little difficulty. More often than not the patient volunteers the information of a pain in the leg and a glance at the temperature chart will almost clinch the diagnosis even before the limb is examined. The elevation of temperature, usually fairly marked; the pain, tenderness, œdema and the pale, somewhat glazed, appearance of the limb, together with the appearance of acute illness, are all relatively unmistakable; and, paradoxically enough, the more acutely ill the patient, the better the prognosis as regards embolism.

Phlebo-thrombosis, on the other hand, is much more insidious in its onset, and its detection is much more difficult. Possibly the most important diagnostic symptom is an increased pulse rate, out of proportion to any temperature elevation, the latter being usually slight. Mahler[8] emphasizes this and describes it as a stepladder pulse.

Certainly any postoperative patient who has, about the 8th to 10th day, a very small rise of temperature (say to 99° only) accompanied by a disproportionate rise of pulse, should be carefully examined for the presence of phlebothrombosis. This examination should include the plantar aspects of the feet, particularly on the lateral side, since, as shown by Neumann[9] the initiating thrombus not infrequently occurs in the plantar veins.

Squeezing the feet, squeezing the calf, and the dorsi-flexion sign of Homans and examination of the femero-iliac veins should be carried out. Quite commonly the only complaint of the patient with phlebo-thrombosis may be "tender toes", and in this respect it is interesting to note that Connor[10] found that 12 out of 22 patients suffering from typhoid fever, and complaining of tender toes, had phlebothrombosis of the leg.

In doubtful cases, or in cases of suspected phlebo-thrombosis where it is of importance to

localize the clot accurately, phlebography should be done. This is of particular importance in the cases of patients who have suffered a small, non-fatal pulmonary infarct, since, as Zink[11] has shown, in 70% of cases of fatal pulmonary embolus, the patient had suffered a previous minor infarction; the result of embolus.

TREATMENT

It is here that one meets with a great diversity of opinion, and bitter controversy between those who uphold anti-coagulant therapy and the advocates of radical surgical procedures. In some centres practically no veins are tied, in others, very large numbers are so dealt with, many even as a purely prophylactic preoperative measure. The Mayo Clinic, and the Boston group might be cited respectively as the most ardent protagonists of these two schools of thought.

Gordon Murray of Toronto has now treated some 2,500 patients with anti-coagulants without a case of fatal pulmonary embolus while under treatment, though it is understood that there have been cases of severe, and even fatal hæmorrhage in this series. However, it is a fact that many eminent authorities are firm believers in the use of heparin, dicoumarol and hirudin in the treatment of these conditions of intravenous thrombosis, as opposed to surgical intervention.

Certainly, though it is generally agreed that anti-coagulants will have no effect upon an already existing thrombus, anti-coagulants should be used in these cases: firstly, as a prophylactic measure, to attempt to forestall thrombosis which might occur in parts other than the one primarily affected; and secondly, to prevent the formation of a propagating thrombus.

The best treatment is, of course, prophylaxis and all, or as many as possible of the predisposing and initiating factors should be eliminated prior to operation. Consideration should also be given to the type of operation proposed; since intestinal and gastric operations, particularly for malignancy, hernias, prostatectomies and amputations of limbs, are some of those notoriously prone to lead to the development of thrombosis. In many clinics the prophylactic use of anti-coagulants is a routine in these types of cases.

Postoperatively, early ambulation has been hailed as possibly the greatest single factor in diminishing the incidence of thrombosis. It is interesting, however, to note that in thoracoplasty operations for pulmonary tuberculosis, where the patient is necessarily bedfast for months before and after operation, the incidence of thrombosis and embolism is extremely low.

Published figures, however, seem to indicate very definitely that early ambulation has materially reduced the morbidity and mortality rates of pulmonary embolism, in postoperative patients in normal general hospital practice. Failing this, pressure bandages applied to the lower extremities, bed exercises, massage, etc., are all advocated by various authorities. One cannot help but feel, however, that once a condition of intravenous clotting has occurred, pinning one's faith exclusively to anti-coagulants is, perhaps, expecting too much of them, and that, particularly if there has been one non-fatal pulmonary infarct, some more active measures to protect the patient from a fatal accident should be initiated. In this connection it is essential that a differential diagnosis between acute thrombo-phlebitis and phlebothrombosis, together with an accurate estimation of the clot level, be made. It is felt that the treatment of the two conditions differs widely, and this is the reason for stressing the differential diagnosis.

Acute thrombo-phlebitis.—As has been previously mentioned, unless in the rather rare event of suppuration, embolus is relatively unlikely in this disease. Consequently, conservative treatment, including the use of anti-coagulants, should be the rule.

Novocaine block of the sympathetic ganglia controlling vaso-constriction in the affected limb is the initial method of choice, though some authors advocate continuous caudal anæsthesia as being superior. Lumbar ganglionectomy may be indicated in resistant cases. Daily blocks will, as a rule, suffice, and it is only rarely that more than two or three will be needed. As a rule, pain is immediately relieved, and the fever and œdema have generally disappeared at the end of 48 hours.

If this procedure is carried out early and the limb is not permitted to remain painful and œdematous for any length of time, leading to great dilatation of the intercellular spaces, the

sequelæ of the painful and œdematous "milk leg" will be rarely encountered. In the event of septic necrosis of the clot—a very unusual occurrence—radical surgery, with ligation of veins well above the affected area, even up to and including the inferior vena cava, must, of necessity be initiated immediately the condition is diagnosed. This may be done alone, or if the condition of the patient will permit, in conjunction with thrombectomy.

One feels that, if possible, prevention of embolus by high ligation should also be accompanied by extirpation of a suppurating thrombus, on the general principle of the removal of any focus of infection.

Phlebo-thrombosis.—Here is an entirely different set of circumstances, requiring an entirely different mode of treatment. The condition is, as has been pointed out, much more insidious in its onset, and more difficult of detection, yet, if one is on the watch for it, is fairly readily recognizable. In this disease, the primary danger is not to the limb, but to the life of the patient. There is no ischæmia, and little vaso-spasm, but there is a great liability to embolization, and treatment must be directed toward the prevention of a catastrophe. Consequently it is felt that the treatment here is early and *radical* surgery.

Phlebography may be done to localize the clot if there is doubt on clinical examination, and the vessel or vessels must be tied well above it. Thrombectomy has been advocated, but it is difficult to justify it, and, in the opinion of the author, is futile. Once one has ligated a vessel above the thrombosed area, one cares little what happens to the distal portion, and, indeed, normally it will become thrombotic, whether or not there has been a pre-existing thrombus present. Under these circumstances it would appear to be "gilding the lily" to remove a primary thrombus, only to have it replaced by a secondary one. It would seem that the only reason for thrombectomy below ligation is the suspicion that the clot might suppurate.

Ligations to combat intravenous thrombosis have become popularized in comparatively recent years, and the boldness of the surgical approach has been remarkable. Surgeons have ascended the venous tree (anatomically, and perhaps Hibernially, speaking) to its trunk. Attacks upon the saphenous vein, and the superficial femoral vein rapidly gave place to ligations of the common femoral and iliac veins, and then the inferior vena cava became a site of election, with gratifying results in the main, and a rather surprising absence of œdema, once sufficient time had elapsed for the establishment of a collateral venous drainage system.

Whilst this appears to be, at first sight, a very radical procedure, clinical experience has demonstrated that, if it be done early, and a sufficient lapse of time be not permitted for the development of marked peri-venous inflammation with exudate, which fills the vascular sheath and directly stimulates arterial spasm, one may be reasonably assured of having, as an end result, a limb, or limbs, without pain or œdema, and with normal function.[12]

The later stage, of course, where a heavy greyish cement-like substance about the vein may be found on exposing it, giving rise to stimulation of perivascular nerves and painful vaso-constriction belongs more properly to the post-phlebitic limb, and as such, is beyond the scope of this paper but it should be emphasized that the development of the post-phlebitic leg, which is an awesome problem for any surgeon to treat, is, more often than not, a sequel to either lack of, or poor treatment of the initial lesion.

SUMMARY

The literature has been reviewed and the underlying pathological lesions in intravenous clotting have been discussed. An attempt has been made to differentiate between two types of intravenous thrombosis and to indicate the rationale of varying the treatment in accordance with the particular type present in any given case.

The question of the use of anti-coagulants alone, as opposed to surgery is discussed.

CONCLUSIONS

1. There are two distinct mechanisms leading to intravenous thrombosis.

2. The results of these are clinically and physiologically so different as to produce what might be termed two allied, but different diseases.

3. The treatment of each is different.

4. Early attempt should be made to determine which type of thrombosis is present

in order that the appropriate treatment may be instituted.

5. The "post-phlebitic leg" can, more often than not, be prevented by early and intelligent treatment of the initial lesion.

The author wishes to acknowledge, with grateful thanks, the work of Miss Mary Gzowski, medical artist to the Montreal General Hospital, in preparing the drawings reproduced.

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Medical Arts Bldg.

ASYMPTOMATIC MINIMAL PULMONARY TUBERCULOSIS: ASSESSMENT OF STATUS: RESULTS OF SUPERVISION AND TREATMENT*

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ASSESSMENT of the status of tuberculous lesions of minimal extent, found on routine or mass x-ray examinations of seemingly well persons, has become an important part of tuberculosis control programs in recent years.

Various investigators, notably Abeles and Pinner[1] and Bobrowitz and Dwork,[2] have stressed the importance, in cases of minimal pulmonary tuberculosis discovered in the course of routine x-ray examinations, of making careful inquiry regarding symptoms. Bobrowitz and Dwork,[2] whose observations were based on a study of approximately 200 cases of minimal pulmonary tuberculosis under treatment in hospital, state that inquiry showed that 81% of their cases had symptoms suggestive of tuberculosis prior to, or at the time they were found to have this disease. Reisner and Downes,[3] on the other hand, state in their report on a study of 469 cases of minimal pulmonary tuberculosis followed in the ambulatory chest clinics of the New York City Department of Health, that 76% had no significant subjective symptoms at the time of initial diagnosis. Most observers seem to agree that physical examinations are of little aid in the recognition of active tuberculous lesions of minimal extent. Kruger, Potter and Jaffin[4] state that serial x-ray pictures proved, in their experience, with both hospitalized and clinic patients with minimal pulmonary tuberculosis, to be an invaluable aid in the recognition of cases of active disease. Reisner and Downes[3] found roentgenological signs an invaluable aid in the evaluation of the clinical significance of lesions of minimal extent in ambulatory patients. Both Kruger and his associates and Reisner and his co-worker note that specimens of sputum and/or gastric contents of only a few of their patients were examined. They state that the demonstration of tubercle bacilli in specimens of sputum or gastric contents constitutes evidence of activity. Reisner and Downes went on record as being of the opinion that more intensive studies, such as repeated cultures and animal inoculations of sputum or gastric contents would, in all probability, as Decker, Ordway and Medlar[5] have pointed out, demonstrate tubercle bacilli in a considerable proportion of cases with minimal tuberculous involvement. Decker, Ordway and Medlar[5] were able, by repeated examinations of sputum and fasting gastric contents, by culture and guinea-pig inoculations, to demonstrate tubercle bacilli in 67 out of 97 patients with clinically active minimal pulmonary tuberculosis hospitalized in Mount McGregor Sanatorium between 1936 and 1941.

More recently Scott[6] reported that tubercle bacilli were found, soon after admission, in specimens of sputum or fasting gastric contents of 46% of the 118 patients with minimal pulmonary tuberculosis who entered the Ninette Sanatorium between 1943 and 1945. Hilleboe,[7] in a publication entitled a *Guide for Disposition of Persons with Abnormal Pulmonary Findings on X-ray Films,* advocates that only persons who give a positive Mantoux reaction and in whose sputum or gastric contents tubercle bacilli can be found, be definitely diagnosed as cases of tuberculosis and given sanatorium care or the equivalent. He indicates that he believes all cases presenting abnormal x-ray

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findings and a positive Mantoux reaction should, until proved otherwise, be regarded as "suspected tuberculosis" and simply kept under observation. Amberson,[8] in a recent editorial in the *American Review of Tuberculosis* went on record as being of the opinion that it is unwise to refrain from giving patients with minimal pulmonary tuberculosis a period of sanatorium care or the equivalent simply because all alleged activity tests are negative and, more particularly, because attempts to demonstrate tubercle bacilli have failed to yield positive results.

In an effort to ascertain the efficacy of certain objective criteria in the assessment of the status of tuberculous lesions discovered in persons who state that they cannot recall having had any complaints in the weeks and months prior to the discovery of their disease, the records of 443 cases of so-called asymptomatic minimal pulmonary tuberculosis were reviewed. The results of this review of the records of a selected number of persons with minimal pulmonary tuberculosis who have been under supervision or treatment for periods ranging from a few weeks to approximately four years will be presented in this paper.

DATA: METHODS

The 443 cases of minimal pulmonary tuberculosis selected for this study represent 31.02% of the total number of cases of minimal pulmonary tuberculosis under supervision or treatment by this Hospital on December 31, 1945. Thirteen of these cases had been under supervision for less than 3 months; 15 had been under supervision for over 4 years. All the cases included in this study were discovered either on mass survey x-ray examination or on routine x-ray examinations of families in which a case of tuberculosis had recently been discovered. Most of the cases had had one or more chest x-ray pictures taken before they reported to the Clinic. In most instances, however, a diagnosis of minimal pulmonary tuberculosis was made only after the patient had had two or three chest x-ray pictures taken several weeks apart and had been seen in the Clinic on two or more occasions. All of the cases included in the present study presented x-ray findings corresponding to those for pulmonary lesions of minimal extent as described in *Diagnostic Standards and Classification of Tuberculosis* (1940).

The procedures followed in the handling of the patients whose records constitute the basis for this study were similar to those followed in the handling of other patients admitted to the Clinic. A history was taken by a nurse trained in history-taking, in each case. This history was reviewed, altered and supplemented, when necessary, by the attending physician. The histories of the 443 cases of minimal pulmonary tuberculosis selected for the present study differed from those of other cases of minimal pulmonary tuberculosis under supervision in that they revealed that none of these patients could recall having had any symptoms referable to the respiratory tract, any undue fatigue or any loss in weight in the weeks or months immediately preceding the discovery of their pulmonary tuberculosis, and all denied cough and expectoration at the time that their pulmonary tuberculosis was diagnosed. Of the 443 patients under consideration 284 (64.11%) were given one or more physical examinations after they first reported for medical advice. An effort was made in each of the cases given a physical examination to determine whether, or not, râles—particularly latent râles—were present. Each of the 443 cases was given a specimen bottle, on one or more occasions and asked, in spite of the fact that he or she denied cough and expectoration, to submit a sample of sputum for examination. Of these patients 161 (36.34%) submitted specimens of sputum considered worthy of examination for tubercle bacilli. Specimens of gastric contents were collected in 70 (15.8%) cases. It may not be amiss to point out here that 18 patients who submitted specimens of sputum considered worthy of examination were, at a later date, given gastric lavages. Most of the samples of sputum were examined by smear alone; a few, however, were concentrated and cultured on Lowenstein's medium. All of the specimens of gastric contents were concentrated and all but those which gave high Gaffky counts—that is, over Gaffky II or III—were cultured. All of the 443 cases included in the present study were followed with serial x-ray pictures. Most of the cases were x-rayed at intervals of two to three months during the first year that they were under supervision. X-ray pictures were, as a rule, taken at longer intervals during subsequent years. In the interpretation of the serial x-ray pictures of these patients

special attention was given to noting whether the abnormal shadows remained stationary or tended either to increase or to decrease in size.

Practically all of the patients included in this study were permitted to continue at work while the status of their pulmonary disease was being assessed. Of the 443 cases 224 (73.14%) were given no special treatment at any time; 75 (16.93%) were, at some time during their period under supervision, advised to take extra rest out of working hours; 45 (9.93%) were advised to give up work and to enter a sanatorium. It may be well to point out here that a goodly proportion of the patients for whom sanatorium care was recommended had, due to the lack of suitable accommodation, to attempt to take the cure in their own homes.

The final step in the review described herein consisted in ascertaining the status, in terms of *Diagnostic Standards* (1940), of all 443 patients, as of December 31st, 1945. This was done to permit of study, in the light of subsequent developments, of the respective rôles of physical findings, x-ray findings and laboratory findings in the assessment of the status of each patient's pulmonary disease on his admission to the clinic as well as to secure information with respect to the results of supervision and treatment of all 443 patients up to December 31, 1945.

ASSESSMENT OF STATUS

(*a*) *Physical findings — râles.* — Râles were found in 20 (7.04%) of the 284 patients in this series who were given physical examinations soon after they reported to the Clinic for advice. Twelve of the 20 patients in whom râles were found were permitted to continue at work. Seven were advised to take extra rest out of working hours. One was advised to follow a sanatorium regimen.

The status of the pulmonary lesions of the 20 patients in whom râles were detected was, as of December 31, 1945, as follows:

Active	0
Quiescent	3
Apparently arrested	3
Arrested	13
Undetermined	1

(*b*) *X-ray findings.*—Serial x-ray pictures of 329 of the 443 patients included in this study showed no change of note as between the first film showing evidence of disease and the most recent film. Serial x-ray pictures of the 443 patients showed signs either of progression or of retrogression of disease. The x-ray findings of an additional 46 patients were such, as of December 31, 1945, that it was deemed advisable to simply classify them as indefinite.

Of the patients whose serial x-ray pictures showed no change of note 252 (76.59%) were permitted to continue at work; 57 (14.28%) were advised to take extra rest; 20 (6.07%) were advised to follow a sanatorium regimen. The status of the pulmonary lesions of the 329 patients whose serial x-ray pictures revealed stationary abnormal shadows was, as of December 31, 1945, as follows:

Active	2
Quiescent	14
Apparently arrested	45
Arrested	261
Apparently cured	4
Undetermined	3

The findings in the 68 patients whose serial x-ray pictures showed evidence of change in abnormal shadows while under supervision may, to advantage, be considered under two headings, namely: (1) those of patients—29 in number—in whose x-ray pictures the abnormal shadows showed a tendency to increase in size; and (2) those of patients—39 in number—in whose x-ray pictures the abnormal shadows tended to decrease in size.

Twelve (41.37%) of the 29 patients whose serial x-ray pictures showed evidence of an increase in size of abnormal shadows were, for one reason or another, allowed to continue at work; 17 (58.62%) were advised to take extra rest or to follow a sanatorium regimen. The status of the pulmonary lesions of these 29 patients, as of December 31, 1945, was as follows:

Active	11
Quiescent	11
Apparently arrested	3
Arrested	4
Apparently cured	0
Undetermined	0

Of the 39 patients whose serial x-ray pictures showed evidence of a decrease in size of abnormal shadows, 23 (58.97%) were permitted to continue at work and 16 (41.02%) were advised to take extra rest or to follow a sanatorium regimen. The status of the pulmo-

nary lesions of these 29 patients, as of December 31, 1945, was as follows:

Active	3
Quiescent	5
Apparently arrested	5
Arrested	24
Apparently cured	0
Undetermined	2

(c) *Laboratory findings — sputum and/or gastric contents.* — Acid-fast bacilli similar morphologically or culturally to *M. tuberculosis* were found in specimens of sputum or gastric contents of 25 (11.73%) of the 213 patients in this series from whom specimens were obtained. Three of the patients in whose specimens of sputum or fasting gastric contents acid-fast bacilli were found were reported, on physical examination, to have had râles. The serial x-ray pictures of these three patients were classified as showing no change of note.

Eleven of the 25 patients in whose specimens of sputum and fasting gastric contents acid-fast bacilli were found were classified, from an x-ray standpoint, as having stationary pictures; 14 were classified, from an x-ray standpoint, as having pictures which showed signs either of progression or of retrogression of their disease. Of these 14, 8 showed signs taken to be indicative of retrogressive changes. It may not be amiss to point out here that specimens of sputum and/or fasting gastric contents of 26 of the 29 patients whose serial x-ray pictures showed signs of progression of their disease were examined for evidence of *M. tuberculosis,* whereas specimens of sputum, of only two-thirds of the 39 patients whose x-ray pictures showed signs of retrogression of their disease, were similarly examined.

Of the 25 patients in whose sputum or fasting gastric contents acid-fast bacilli similar to *M. tuberculosis* were found, 8 were, for one reason or another, allowed to continue at work; 7 were advised to take extra rest out of working hours; and 10 were advised to follow a sanatorium regimen.

The status of the pulmonary lesions of these 25 patients, as of December 31, 1945, was as follows:

Active	9
Quiescent	5
Apparently arrested	3
Arrested	8
Apparently cured	0

RESULTS OF TREATMENT

The simplest method to employ in attempting to ascertain the results of using physical, x-ray and laboratory findings as described herein, in the assessment of the status of tuberculous lesions of minimal extent in the lungs of persons who are asymptomatic, and of treating patients whose disease is considered active, in the manner herein set forth, appears to be to record the status of the pulmonary lesions of all of the patients under consideration at some fixed date. The status of the pulmonary lesions of the 443 cases included in this study, as of December 31, 1945, was as follows:

Active	18
Quiescent	32
Apparently arrested	57
Arrested	302
Apparently cured	4
Undetermined	30

Examination of the foregoing table shows that, as of December 31, 1945, 363 (81.94%) of the 443 patients included in this study had apparently arrested, arrested, or apparently cured disease and that only 50 (11.28%) of the entire group had active or quiescent disease. This appears to be a reasonably good result, if allowance is made for the following facts: (1) that 30 patients (6.76%) had been under supervision too short a time as of December 31, 1945 to warrant a statement as to the status of their pulmonary disease. (2) That most of the patients had to be permitted to continue at work until it became clearly evident that their disease was active. (3) That a few patients who were known to have active tuberculosis were, through force of circumstances, obliged to continue to work and to limit their cures to whatever rest they could secure out of working hours. It leaves much, however, to be desired, in that 50 persons who were found on routine x-ray examination to have minimal pulmonary tuberculosis and were promptly referred to the clinic, still had active or probably active disease six months or more after they first reported for medical advice.

DISCUSSION

The foregoing observations seem clearly to indicate that a significant proportion of persons, found as a result of routine or mass x-ray examination to have minimal pulmonary tuberculosis, can be expected to deny symp-

toms even remotely suggestive of this disease and that a significant percentage of those who deny symptoms can be expected to have tuberculosis in an active form at or about the time their pulmonary disease is discovered. The cases of asymptomatic minimal pulmonary tuberculosis which form the basis for the present study constituted 31.02% of the total number of cases of minimal pulmonary tuberculosis under supervision and treatment by this Hospital on December 31, 1945. This is a much lower percentage of asymptomatic cases than that reported by Reisner and Downes[3] in their series of 469 cases of minimal pulmonary tuberculosis. The latter observers reported, it will be recalled, that 75% of their cases had no significant symptoms at the time of initial diagnosis. The fact that a goodly percentage of the cases of asymptomatic minimal pulmonary tuberculosis included in this study were found to have active disease within a few weeks of the time they first came under supervision, seems to indicate that careful inquiry regarding symptoms is not as valuable a guide in the recognition of active disease as Bobrowitz's and Dwork's[2] observations suggest might be the case.

The study lends weight to the now widely held view that physical examinations, including special efforts to detect latent râles, are of little or no value in the assessment of the status of minimal pulmonary tuberculosis in seemingly well persons.

The observations recorded herein demonstrate that serial x-ray pictures are of inestimable value in the assessment of the status of asymptomatic minimal pulmonary tuberculosis. The criteria employed in the interpretation of the serial x-ray pictures of the patients included in this study differed from those used by Reisner and Downes[3] and by Kruger, Potter and Jaffin.[4] The criteria employed herein had, for the most part, to do with lack of change, or changes, in the size of the abnormal shadows rather than with the character of the abnormal shadows at the time the disease was discovered, or at some subsequent date. The fact that only 16 of the 329 patients, whose serial x-ray pictures showed no significant change in the size of the abnormal shadows during their periods of supervision and treatment, were classified as having active or quiescent disease on December 31, 1945, seems to indicate that stationary abnormal shadows constitute a reasonably accurate guide to the status of tuberculous lesions of minimal extent discovered on routine x-ray examination of seemingly well persons. The fact that all of the 329 patients whose serial x-ray pictures showed no change of note could not be classified as having apparently arrested, arrested, or apparently cured disease at the end of the period under review, indicates that stationary abnormal shadows cannot always be taken to mean that the disease which they depict is inactive. One other observation with respect to the serial x-ray pictures of the patients included in this study seems to deserve special comment. The findings recorded herein suggest that patients in whose serial x-ray pictures the abnormal shadows show a tendency to decrease in size are almost as certain to have active lesions as the patients in whose x-ray pictures the shadows show a tendency to increase in size. Comparison of the status, as of December 31, 1945, of patients in whose serial films the abnormal shadows tended to decrease, and of those in whose films the shadows tended to increase, seems clearly to indicate that the former group of patients is likely to do better than the latter group in so far as arrest of their disease is concerned.

This study demonstrates that examinations of specimens of sputum or gastric contents for tubercle bacilli can, even in patients who present x-ray evidence of only minimal pulmonary tuberculosis and who deny cough and expectoration, be an invaluable aid in the assessment of the status of lesions. Specimens submitted by, or collected from, 213 of the patients whose findings form the basis for this study, were examined. Acid-fast bacilli, morphologically or culturally similar to *M. tuberculosis* were found in specimens from 25 of these 213 patients. The patients in whose sputum or gastric contents tubercle bacilli were found were not a selected group, except in the case of those whose serial x-ray pictures showed signs of change in the size of the abnormal shadows. It seems reasonable, therefore, to expect that tubercle bacilli might have been found in specimens submitted by, or obtained from, more patients if it had been possible to obtain more specimens and, perhaps, to make more examinations for tubercle bacilli by means of cultures. The findings recorded herein show that the serial

x-ray pictures of 11 of the 25 patients, in whose sputum or gastric contents tubercle bacilli were found, presented stationary abnormal shadows. This seems to be a clear indication that examinations of specimens of sputum submitted by, or collected from, patients with so-called asymptomatic minimal pulmonary tuberculosis must, as several recent investigators' reports tend to show, be given much more attention in the assessment of the status of tuberculous lesions of minimal extent than has been the case in the past.

Finally, the fact that approximately 82% of the 443 cases of asymptomatic minimal pulmonary tuberculosis whose findings form the basis for this study, could be classified as having apparently arrested, arrested, or apparently cured disease on December 31, 1945, in spite of the fact that lack of sanatoria facilities made it impossible to give approximately half of the known cases of active tuberculosis the kind of treatment which they really should have been given, seems to add weight to the view that the prognosis is good in most cases of minimal pulmonary tuberculosis found on routine x-ray examination of seemingly well persons. It is regrettable that slightly over 11% of the cases included in this study had to be classified as having active or quiescent disease at the end of the period of observation. Such evidence as is available at present suggests that adequate sanatoria facilities for patients found to have active lesions of minimal extent may be expected materially to reduce the numbers and the percentage of those who fail to arrest their disease.

SUMMARY

The findings of 443 persons, who were found on routine or mass x-ray examinations to show signs clearly indicative of minimal pulmonary tuberculosis and who could not recall having had any complaints suggestive of tuberculosis in the weeks and months prior to the discovery of their disease, have been reviewed in an effort to assess the efficacy of certain objective criteria, namely: (1) Physical examinations, including special efforts to detect latent râles; (2) serial x-ray pictures, and (3) examinations of specimens of sputum or gastric contents for acid-fast bacilli morphologically and culturally similar to *M. tuberculosis*.

This review has demonstrated that physical examinations, including a search for latent râles, are of little or no value in the assessment of the status of tuberculous lesions of minimal extent. It has, on the other hand, shown that serial x-ray pictures, and examinations of specimens of sputum or gastric contents obtained from patients who deny cough and expectoration, are invaluable aids in the assessment of the status of minimal pulmonary tuberculosis found on routine x-ray examination of seemingly well persons.

The findings recorded herein seem clearly to indicate that bacteriological examinations of specimens of sputum submitted by, or collected from, patients with so-called asymptomatic minimal pulmonary tuberculosis should, as several recent investigators' reports tend to show, be given more attention in assessment of the status of tuberculous lesions of minimal extent than has been the case in the past.

Classification of the status of the tuberculous lesions of the 443 patients whose findings constitute the basis for this study shows that on December 31, 1945, the lesions in approximately 82% were either in an apparently arrested, an arrested or an apparently cured state. This observation seems to indicate that the criteria used in the assessment of the status of the lesions of these patients at the time that their disease was discovered yielded reasonably accurate results. It is regrettable that approximately 11% of the patients included in this study proved to have active or quiescent lesions at the end of the period of observation. Such evidence as is available at present suggests that lack of adequate sanatoria facilities for the treatment of persons found to have active tuberculous lesions was a factor in preventing these patients from bringing their disease under control.

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Atheism is rather in the life than in the heart of man.—Bacon.

ACUTE PANCREATIC ŒDEMA

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THE writer's interest in the subject of acute pancreatic œdema (synonyms: acute interstitial pancreatitis, acute transient pancreatitis or subacute pancreatitis) was aroused by the following case.

Mrs. A.B., aged 43, weight 250 pounds, married and the mother of eight children, was seen at her home January 27, 1948, with severe epigastric and right subcostal pain of eight hours' duration. The pain was described variously as boring and colicky and was referred through to the back. There had been nausea and vomiting prior to my arrival. There had apparently been minor attacks of epigastric pain for the previous three weeks. The urine was said to be dark but the stools were not clay coloured. There was a history of abdominal pain, diagnosed as gall bladder trouble fifteen years before. There had also been two operations, one for sterilization and one for removal of a fat pannus. The temperature was not elevated and the pulse was 66 per minute. There was tenderness in the epigastric and right subcostal area, but little splinting or rigidity. No masses were palpable. The lower abdomen was quite soft. She was considered to be having a gall stone colic, probably from a common duct stone, and was given morphine grains ¼ and atropine gr. 1/150 hypodermically.

Little or no relief was obtained and the patient was admitted to hospital late the same afternoon. On admission, she was somewhat cyanosed and was obviously in severe pain: temperature 97.4°, pulse 80 and the blood pressure 165/115. The morphine was repeated and local heat applied. The pain gradually abated but during the next three or four days she had repeated attacks of a minor character, with nausea and vomiting on two occasions, and complained of pain in the left upper quadrant now rather than the right. The temperature ranged up to 101.8° F. Urinalysis on admission showed +1 sugar and a trace of bile. Urinary diastase on the morning of January 28 showed 128 units (normal 6 to 32 units).

An electrocardiogram recorded only left axis deviation. A cholecystogram was reported as revealing a non-functional gall bladder with no evident stones.

The symptoms abated, fever subsided and operation was performed February 6. A gall bladder, not at all acutely inflamed, but containing many small stones was seen. The common duct was dilated and a calculus was palpable in its lower end. The pancreas was the seat of a firm, lobulated enlargement without evidence of fat necrosis or hæmorrhage. Cholecystectomy, and choledochostomy (after removal of the palpable stone), were performed. Culture of the bile subsequently exhibited colon organisms. The patient made a good recovery, the T-tube being removed on the sixth post-operative day. The tract drained only a small quantity of bile for 24 hours and has since remained healed.

The pathological report of the gall bladder was "Chronic productive cholecystitis with cholelithiasis".

When seen on March 2, the patient had only a trace of sugar and was entirely free from pain.

The existence of a type of mild, transient pancreatitis involving the whole or part of the gland has been remarked on by many surgeons, being usually seen at operations performed for disease of the biliary system. Halsted[1] noted it in 1890 when he operated on a patient for acute obstruction and found only a hard, indurated pancreas. He closed the abdomen and recovery ensued. It was further suggested by Archibald,[2] in 1913, when he reported the case of a young man who had several acute but transient attacks of agonizing epigastric pain with localized epigastric tenderness and who, at operation, revealed a diffusely swollen pancreas without fat necrosis or hæmorrhage. The abdomen was closed and the patient recovered. The belief was expressed that many of the cases of acute epigastric pain disappearing within a few hours or more, and ordinarily diagnosed as of gastric or gall stone origin, were really examples of this mild form of pancreatitis.

In 1922 Zoepffel[3] stimulated clinical interest when he reported four cases in which, at operation, the pancreas was found to be swollen and œdematous but where no hæmorrhage or necrosis was present. In two of them there was some fat necrosis and in two a stone was present at the papilla of Vater. Microscopic examination of a biopsy in two cases revealed no evident necrosis. Analyzing 2,137 cases of pancreatitis in 1927, Schmieden and Sebening[4] reported 44% as showing only œdema and fat necrosis without hæmorrhage or necrosis of the gland. Elman,[5] in 1933, surveyed the literature on the subject and summarized 37 cases of acute interstitial pancreatitis in a detailed analysis. It was his definite opinion that the cases represented a distant pathological entity and were not merely an early stage in the development of pancreatic necrosis. Other authorities, however, including Bockus,[6] feel that œdema of the pancreas represents, to quote, "only a form of acute inflammation in which the causative agent is less fulminant or is active only transiently!"

The pathogenesis of acute interstitial pancreatitis is, of course, bound up with that of acute pancreatitis in general. Rich and Duff in 1936[7] confirmed that acute pancreatitis could be produced by the injection of practically any irritant, from bile and duodenal secretion, to weak acids or alkalis, calcium chloride, formaldehyde and formic acid, etc., into the pancreatic duct. They said that the resulting pancreatitis was due to the rupture of acini and ductules and:

"If the cells at the sites of the ruptures are damaged by the irritant or spread further apart by inflammatory œdema, the openings will not become promptly sealed up, as occurs after the injection of bland fluids, and if pancreatic juice of sufficent tryptic potency escapes and comes into contact with large arteries, vascular necrosis with resulting hæmorrhage, thrombosis, tearing of the gland and infarction will produce the picture of hæmorrhagic pancreatitis!"

Chisholm and Seibel,[8] using the experimental method described by Rich and Duff,[7] injected freshly aspirated bile from the gall bladder into the duct of Santorini in dogs. When less than 2 c.c. of bile at less than 18 cm. of water pressure was injected slowly, the ductal tree of the pancreas was filled but not ruptured. Acute œdema of the pancreas was seen to develop within a few minutes. The lobules were seen separated by a transparent transudate. Microscopically, this showed some leucocytes but mainly interacinar œdema. When 2 to 11 c.c. of bile were injected at a pressure greater than 18 cm. the ductal tree was ruptured, the intralobular œdema became bile stained and in a short time small petechiæ appeared in the acinar substance. Microscopic examination of pancreatic sections now showed extravasated erythrocytes, leucocytes, fibrin and granular debris. Several hours after injection this type of pancreas showed fat necrosis on the gland and adjacent peritoneum. Using ten acute experiments in a 12 hour period, they showed that when the smaller amount was injected and the ducts not ruptured, serum amylase levels rose rapidly but tended to return to nearly normal by the end of that period. At autopsy these dogs showed only œdema of the pancreas. On the other hand, where the larger amounts were injected under the greater pressure, and where necrosis was evident later at autopsy, the serum amylase levels continued to rise even at the end of the period.

Similarly, following a series over a 7 day period, they found that the cases of pancreatic œdema had amylase levels which rose rapidly but which soon declined toward normal. The cases which later showed pancreatic necrosis, however, had higher enzyme levels which only slowly subsided.

It was established by Rich and Duff[7] that trypsin freed by the rupture of ducts and acini, and activated by calcium ions of the tissue fluids, and in contact with large pancreatic vessels, could produce hæmorrhagic pancreatitis, this without the presence of enterokinase which ordinarily activates trypsinogen. Ingestion of alcohol or a heavy meal was found to increase the tryptic and, therefore, the destructive power of the juice, while conversely starvation produced a juice of lesser potency. Hence the occurrence of acute hæmorrhagic pancreatitis following on a heavy meal or an alcoholic bout, and perhaps the milder pancreatic œdema may be associated at times with a juice of lower tryptic potency.

The mode of activation of the pancreatic juice clinically is still in much dispute. The "common channel" theory whereby a reflux of bile, often infected, takes place into the pancreas is the obvious answer, but it does not explain a good many cases. Opie,[9] in 100 routine autopsies, found a common ampullary system in 89. Perhaps the consensus of numerous surveys suggests that in only 65 to 75% of cases do the bile and pancreatic ducts communicate. In 40 to 70% of pancreatitis cases, gall stones have been found in the gall bladder but in less than 5% (Schmieden and Sebening[4]) has a stone been found at the ampulla of Vater.

Archibald[14] proposed and strongly supported the theory of spasm of the sphincter of Oddi to explain those cases where no obstruction by stone or other cause was present. Evidence which supports the "common channel" theory is the fact that pancreatic ferments have been found in the gall bladder in many cases of pancreatitis and that the gland itself in pancreatitis often has a yellowish or greenish colour (7 out of 37 of Elman's[5] cases showed this). Rich and Duff,[7] on the other hand, advocate metaplasia of the pancreatic ducts as the obstructing factor in most cases, and this would certainly explain some cases of localized pancreatitis and others where no connection with the biliary system is evident. A few cases have definitely been found to be due to obstruction of the pancreatic duct by tumour, stricture, worm or duodenal diverticulum. At any rate, it would seem that the common factors in all cases are the presence of obstruction and the action of trypsin.

Clinically, interstitial pancreatitis has important features. A majority of such cases have had recurrent attacks of pain over weeks or years, often with complete relief between attacks. Elman's[5] series contained 24 (of 37) cases with previous attacks. In many of them no gall bladder lesion is found. Indeed, in one

case reported by Amadon[10] where there had been attacks of what were considered to be typical biliary colic, the pancreas was found to be indurated but no gall bladder could be found at all.

Pain, the main symptom, may be prostrating and paralyzing like peptic ulcer perforation, or colicky with distension like an obstruction. It was severe enough that, in Elman's[5] series, 13 out of 30 cases in which it was recorded, were operated on within 24 hours. In most cases the pain is epigastric but it may be fairly generalized, or right or left subcostal. It may go through to the back and occasionally to the shoulder. The sensation of an epigastric band has also been mentioned. Shock and cyanosis are usually less than in hæmorrhagic pancreatitis, but may occur. Nausea and vomiting are common. Local tenderness is generally epigastric but may be right or left subcostal. Glycosuria is a fairly common finding, and there may be bile in the urine. Enzyme studies reveal a raised serum or urinary diastase. It is interesting to note that in Elman's[5] cases, 6 were diagnosed as intestinal obstruction, 7 as cholecystitis, 9 as perforated ulcer, 1 as appendicitis and one as a perforated gall bladder.

Pathological findings of Elman's[5] cases revealed œdema of the pancreas and peritoneum over it, often spreading into the mesocolon and adjacent duodenum. The parenchyma of the gland was swollen and often hard and indurated. Microscopically there was no evident necrosis but marked infiltration of acute inflammatory cells into the interstitial tissues between lobules as well as acini, often with evidence of œdema. In 16 of 35 cases reported the gall bladder was diseased, with or without stones. Fat necrosis was evident in 15 cases without evidence of gland necrosis.

The treatment is undoubtedly conservative in the acute phase. Shock must be treated primarily where it is found. Probably the only cases subjected to emergency operation should be those where perforation of a viscus cannot be definitely excluded even with the aid of the diastase tests. In this regard McKorkle and Goldman[15] state that an increased serum amylase is specific for acute pancreatitis with the occasional exceptions of acute parotitis and certain kidney diseases with impaired excretion. They state that in 43 cases of acute pancreatitis it was elevated in every case, and that in acute abdominal disease, "when the amylase test is done within the first 2 or 3 days of the acute illness, a low or normal reading is of considerable value in eliminating the likelihood of acute pancreatitis". It has been established that the result of repeated tests parallels closely the clinical course of the condition. An exacerbation of pain is often marked by a rise in the diastase level, and an improvement by a lowered reading. (The exception to this is in the end stages of pancreatic necrosis when the gland is all but spent and the diastase is no longer being excreted). As we have seen, the diastase level tends to fall rapidly in pancreatic œdema and more slowly in necrosis. The value of urinary over serum diastase estimations is that the former remains elevated somewhat longer and therefore may allow of recognition of the condition after the serum diastase has returned to normal.

Some clinicians have utilized serum lipase estimations as an alternative or coincidentally. It was found by Elman[16] to be elevated in 17 out of 20 proved cases of acute pancreatitis. In such emergency operations, cholecystostomy is probably the treatment of choice in most cases. Incision and drainage of the pancreas are no longer considered necessary except where suppuration has taken place and an obvious abscess is present. In those cases which subside under conservative management, investigation and treatment of biliary disease when it is present are urgently called for.

McGravach[11] reported obtaining a history of previous milder attacks of pancreatitis in 7 of 9 cases who died of acute pancreatic necrosis and upon whom autopsies were done. Similarly, Stetten[12] reported recurrent attacks in 12 of 14 cases studied. It is felt also that this milder type of pancreatitis may be a forerunner of pseudocysts, abscess formation and perhaps, chronic pancreatitis. There is, as well, a real danger of diabetes developing in a gland recurrently attacked. It is estimated that 2 to 3% of acute pancreatitis cases become diabetics. It would therefore, seem highly expedient to attempt to prevent further attacks if at all possible. Prophylactically, avoidance of alcohol, large rich meals and control of obesity are to be desired. Small, frequent, low-fat feedings should be prescribed. Where radiological investigation of the biliary tract is suggestive, operation should be carried out.

A diseased gall bladder, with or without stones, should be removed and, in every case, the common duct should be explored and drained.

It would seem logical that prolonged drainage of the common duct, or alternatively, choledochoduodenostomy might be of value in those recurrent cases where no obstruction or biliary disease was found, to prevent development of the chronic condition. For those recurrent cases with intractable pain where no obvious cause is evident, or where the pain persists after operation, De Takats[13] has advocated a diagnostic paravertebral block, from 6th to 10th dorsal, and if this gives relief, states that unilateral splanchnicectomy and ganglionectomy should be done.

SUMMARY

A case of acute pancreatic œdema with chronic cholelithiasis and a stone in the common duct is presented.

A survey is given of some of the literature on the subject.

A plea is made for the recognition of this mild type of pancreatitis, whether as a separate entity or as a stage in acute hæmorrhagic pancreatitis is immaterial, and for investigation of the biliary tract in every case. When biliary disease is present the common duct should always be explored.

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Definition of words has been commonly called a mere exercise of grammarians; but when we come to consider the innumerable evils men have inflicted on each other from mistaking the meaning of words, the exercise of definition certainly begins to assume rather a more dignified aspect.—Sydney Smith.

CONGENITAL ATRESIA OF THE NASOLACHRYMAL DUCT*

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THERE is perhaps no branch of biology which plays so important a rôle in heredity as does the science of ophthalmology. Of the variety of conditions that may present themselves, not the least important is obstruction of the lachrymal passages. A series of cases of congenital dacryostenosis recurring in four generations is herein presented.

A knowledge of embryology is essential to appreciate fully the developmental anomalies. Mann,[1] in describing the development of the lachrymal passages, states that the sac and nasal duct originate as a cord of buried epithelium formed by the maxillary process. The tucked-in epithelium separates from the surface and by budding from its upper end maps out the canaliculi and puncta. The caruncle is developed by a snaring-off of the part of the lower lid fold medial to the opening of the inferior punctum. All the passages are at first blocked out in solid epithelium, and only canalize just before birth. The last part to open is the lower end of the nasal duct which is at times closed by a thin membrane even at birth. Defects of three types may be found. Either the passages do not develop, or the epithelial buds from them will pursue an abnormal course or the apparatus will fail to canalize. All these conditions are known to occur. Gunn[2] cites the possibility of the cartilage being at fault. At birth the cartilages representing the lachrymal, superior maxillary, and inferior turbinate bones bound the duct more or less completely, being thickest toward the lower end of the duct where it is crossed by the turbinate. In the fetus there are cartilages which later disappear, and are not permanently represented by any bone in the adult skull. One such is a layer that connects the cartilaginous ethmoid above with the lateral cartilage of the nose below. Any abnormal growth or persistence of the cartilages might therefore block the duct.

Much has been written on the various affections of the lachrymal apparatus, but surprisingly enough only a scant amount of literature is available dealing with congenital dacryostenosis. Gates[3] in his volume refers to two Russian families, described by Poljak and Popova,[4] with inflammation of the tear sac. In one family there were nine members in four generations; in the other three cases in three generations. Schnyder[5] described a family with cases of tear duct defect in three generations. Macklin[6] has noted the occurrence of a case in mother and child. Three cases in a family of five and the manifestation of the anomaly in the paternal grandfather were described by Emmert.[7]

* Read at the meeting of the Section of Ophthamology, Academy of Medicine, Toronto, November 10, 1947.

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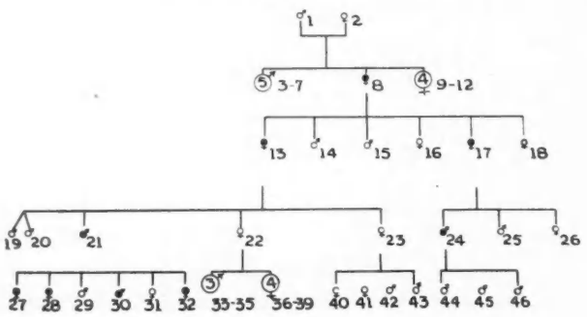
The present series of cases of congenital atresia has occurred in four generations with nine members affected (Fig. 1). These families have lived mainly in the Province of Ontario, and four members (V, 27, 28, 30, 32) were admitted as patients to the Hospital for Sick Children in Toronto. Although six of the affected persons were females, this distribution between the sexes is not statistically significant.

Fig. 1

Pedigree over five generations of the occurrence of Congenital Atresia of the Nasolacrimal Duct



CASE REPORT

The case now presented was a seven-year old girl (V-27), who was first admitted for treatment with a diagnosis of chronic bilateral conjunctivitis. The history revealed that tearing had been present since birth with periodic recurrent acute attacks of conjunctival inflammation. The mother was an American Indian, while the father was descended from English ancestors, his great grandparents having come to Canada as immigrants. Three siblings of V-27 suffered from a similar affection as well as her father (IV 21) who has had the malady all his life. His offspring consisted of six children, five living and one dead. The deceased child (V 31), stillborn at term, had a marked deformity of the head, the nature of which was not determined. Of the five remaining children only the older boy (V 29) showed no malformations. Examination of the eldest daughter (V 27) revealed bilateral dacryostenosis, absence of the superior puncta and an internal strabismus. The second (V 28) and third (V 32) daughters showed similar manifestations without the squint. Developmental anomalies of the same nature could be demonstrated in the youngest boy (V 30). In addition there was noted a horn-like outgrowth which deformed the distal phalanx of his left fifth finger; this had been present since birth. Mentally all the children were alert and had shown satisfactory progress at school. It is interesting to note that the eldest daughter exhibited artistic tendencies well above the average.

The father had two sisters and twin brothers, the twins (IV 19, 20) were born one month prematurely and had died at the age of two weeks. It was not known whether any congenital anomalies existed in their case. The oldest sister (IV 22) was not examined, but as far as could be determined she had no complaints in reference to her eyes or any other part of the body. Her offspring consisted of three boys (V 33, 34, 35) and four girls (V 36, 37, 38, 39)—all normal. The youngest sister (IV 23) bore four children, two girls (V 40, 41) and two boys (V 42, 43), but here again no anomalies could be demonstrated.

Related to the father (IV 21) were three first cousins, two males (IV 24, 25) and a female (IV 26). The latter died of scarlet fever at an early age and from all reports was not abnormal. Information received in respect to the youngest cousin (IV 25) revealed no usual features with the exception that he manifested difficulty in distinguishing colours. The oldest cousin (IV 24) stated that he had tearing of both eyes for as long as he could remember, especially troublesome with cold or windy weather. When examined it was noted that he had a bilateral ptosis as well. The drooping of the lids was stated to have increased in recent years. His three boys (V 44, 45, 46) upon examination had no symptoms or eye abnormalities.

The mothers (III 13, 17) of the two affected cousins came from a family of six; two of these had suffered from tearing all their lives. The other four members, two males (III 14, 15) and two females (III 16, 18) were said to be normal. As five in this generation were deceased, the survivor (III 15), an elderly man gave no history of tearing and when examined showed no abnormalities.

Little information could be obtained about the second generation illustrated in Fig. 1. This family consisted of five males (II 3, 4, 5, 6, 7) and five females (II 8, 9, 10, 11, 12). The only affected member was female (II 8), the mother of the third generation. She had suffered tearing from both eyes all her life. The other descendants of this second generation could not be traced. The parents (I 1, 2) of these ten children had come from England but were not affected.

COMMENT

A genealogic tree illustrating the occurrence of congenital dacryostenosis over five generatoins has been presented. Of 46 members 9 were afflicted with the anomaly, three males and six females. It is evident from this pedigree that this rare anomaly was inherited as a dominant factor. It is interesting to note that the anomaly first appeared in generation II, where only one individual out of ten was affected. It would seem that the defect arose as a mutation in individual II 8, who passed the defect on to two of her six children in generation III. In the following generation IV, two out of eight members inherited the defect. But in generation V the offspring of an American Indian and English union showed a much more frequent incidence with four out of six siblings affected.

This variation among the generations in the frequency of occurrence may on the one hand be due entirely to chance sampling, but on the other hand it raises the question of the penetrance of the dominant factor. It is now established that a few dominant genes produce their trait in every individual carrying the gene. The best example of dominant genes which have complete penetrance are the dominant genes for the blood group series O, A, B, and AB. Other dominant genes have incomplete penetrance, varying from 90%, 60%, 10% to a fraction of a per cent. The reduced penetrance is due to inhibiting effects of other inherited factors or of environmental factors.

The following table shows the expected and observed numbers of offspring in generations III to V.

TABLE I.

TABLE SHOWING THE EXPECTED AND OBSERVED NUMBERS OF OFFSPRING IN GENERATIONS III TO V OF A FAMILY WITH CONGENITAL ATRESIA OF THE NASOLACHRYMAL DUCT

	No. of offspring	*Expected No. affected*	*Observed No. affected*	*% of penetrance observed*
Generation III	6	3	2	
Generation IV	8	4	2	
Totals....	14	7	4	57
Generation V	6	3	4	133

The complete penetrance of the dominant gene in generation V may be due to factors of incompatibility resulting from the mixed American Indian and English cross. This incompatibility may lie in the inherited differences for the growth of the bridge of the nose and surrounding skeletal parts. The combined patterns of growth of the Indian and English having led to an incompatibility which allowed the easier penetrance of the abnormality of the nasolachrymal duct.

On the other hand, the increased penetrance may be the result of a maternal and fetal incompatibility. Recent research has suggested that such incompatibility may result from differences in racial genetic make-up. If a maternal and fetal incompatibility existed, then the developing embryo would find itself in an unfavourable uterine environment in which the dominant gene could more successfully penetrate.

SUMMARY

1. A genealogical tree showing the occurrence of congenital atresia of the nasolachrymal duct over five generations is reported. A total of 46 members is recorded, of which 9 were found to be afflicted with the anomaly. This pedigree shows an inheritance of the dominant nature.

2. The question of penetrance of the dominant factor is commented upon.

3. A table illustrating the expected and observed numbers of offspring in generations III to V is presented.

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PRESENT STATUS OF GOLD THERAPY IN RHEUMATOID ARTHRITIS*

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THE recent conference in Ottawa, under the chairmanship of the Hon. Paul Martin, Minister of the Department of National Health and Welfare, which established the Canadian Arthritis and Rheumatism Society, again emphasized that rheumatoid arthritis is a major public health problem. Although the actual cause and specific cure of this disease are still unknown, it is exciting to contemplate and speculate on the effect of the inauguration of a new era in science, because the atomic age, with consequent increased activity in physics, biochemistry, bacteriology and pathology, should inevitably make an important contribution, before many years, to our knowledge of rheumatoid arthritis.

However, even if there is no specific treatment, no trained observer will deny that a combination of different measures is, in many cases, most helpful. Syphilis was treated with mercury for years before the discovery of the *Treponema pallidum*, and rickets with cod-liver oil before we had any idea of vitamin D and its various constituents. So it is felt today by many physicians that injections of gold salts should be used for the treatment of rheumatoid arthritis in combination with general measures of rest both mental and physical (including local rest of the involved joints by temporary bivalve splinting when necessary), adequate diet, physical medicine (physio- and occupational therapy), eradication of obvious foci of infection as a general health measure, and symptomatic care as indicated, such as analgesics, blood transfusions and iron medications.

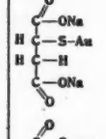
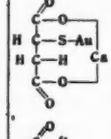
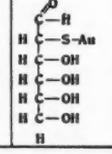
* Read before a meeting of the Rheumatology Section of the Montreal Medico-Chirurgical Society on April 15, 1948.

Marked difference of opinion still exists among rheumatologists as to the value of vaccines, vitamin D concentrate, bee and cobra venoms, etc., *ad infinitum*. At present, most of the controversy concerns the use of vitamin D concentrate in massive dosage and gold therapy. Vitamin D concentrate is slowly making its exit from the therapeutic stage, and there are practically no unprejudiced, competent rheumatologists who advocate its use today. Moreover, the majority who have employed it under careful clinical conditions condemn it as useless and dangerous to the patient. Gold salts, on the other hand, while not considered as a panacea, are regarded with some favour by physicians interested in rheumatoid arthritis all over the world with the possible exception of Russia. It is on this account that it seemed worth while to review this subject briefly and to outline the present method of procedure. As a preliminary to the discussion, it should be recalled that even so-called specific drugs are seldom effective in 100% of cases, as we have learned with arsenic, various sulfa preparations, penicillin, streptomycin, and also, of course, with the injection of gold salts.

A historical review of the use of gold as a therapeutic agent would be the subject for another paper. Suffice to say here that in the eighth century it was recommended as a panacea, and in 1500 in a compound with mercury, advocated by Paracelsus as an elixir of life. However, the use of gold salts in the treatment of rheumatoid arthritis is largely due to the influence of Feldt[1] in Germany and Forestier[2] in France. Feldt showed experimentally in animals that certain gold salts possessed antibacterial powers, and Forestier employed gold salts on a large scale in the treatment of rheumatoid arthritis. From 1930, the use of gold salts in the treatment of this disease has gradually increased, and they are now very extensively employed in Europe and America. Until about 1916, certain preparations of gold salts were injected intravenously and in large doses, with consequent dangerous toxic manifestations and occasional fatalities. Today, gold salts are invariably injected intramuscularly and in much smaller amounts. Numerous articles on their value in human beings have been published, and in Great Britain, the names of Hartfall and Fraser should be especially mentioned, while in the United States, such authorities as Freyberg, Dawson, Boots, Cecil, Hartung and others have reported favourably on their use.

Various salts of gold have been employed. Table I, which is taken from Freyberg,[1] includes the two most commonly used, myochrysine and solganol-B. oleosum, both of which are soluble gold salts and contain about 50% of gold. Colloidal preparations and insoluble organic and inorganic preparations are, generally speaking, unsatisfactory and uncertain in their action, because of irregularity of absorption of insoluble preparations and phagocytosis by the reticulo-endothelium,

Table I
GOLD PREPARATIONS STUDIED

Chemical name	Trade name	Formula	Solubility in water	Physical state	Gold content, per cent
Gold sulfide	Aurol sulfide	Au_2S_3	-	Colloidal suspension	87
Gold sodium thiosulfate		$Na_3Au(S_2O_3)_2$	+	Aqueous solution	37
Gold sodium thiomalate	Myochrysine		+	Aqueous solution	50
Gold calcium thiomalate	Calcium auro-thiomalate		-	Oil suspension	50
Gold thioglucose	Solganol-B oleosum		+	Oil suspension	50

especially in the liver and spleen, of colloidal ones. Experimental work in animals by Feldt has demonstrated that certain preparations of gold possess definite anti-bacterial properties against certain strains of streptococci. Sabin,[4] Freyberg,[5] and others have shown that gold preparations are definitely effective in arthritis produced experimentally in mice by means of pleuro-pneumonia-like organisms. Dawson and Hobby[6] have shown that gold salts have active chemotherapeutic properties against hæmolytic streptococcal infections in mice, comparable to those obtained with the sulfonamides. However, the disconcerting fact remains that in man no organism has yet been found to be of etiological significance in rheumatoid arthritis.

The consensus of reliable, experienced rheumatologists seems to be that gold, in some manner, arrests or lessens the inflammatory

process in many cases of rheumatoid arthritis, but it will not repair damaged cartilage or bone, or reconstruct joints, and it will not rehabilitate the arthritic derelict who has crippling deformities and whose arthritis has been burned out after years of activity of the process. The patient who derives the greatest benefit from proper chrysotherapy is the one with active rheumatoid arthritis, preferably in the early stages of the disease. Although gold therapy is most effective in the early stages, it frequently relieves pain and stiffness even in advanced ones and is worthy of a trial for such patients if there is still evidence that an active process is present. The physician should not use gold salts in any form of rheumatic disease other than rheumatoid arthritis. There is no indication for gold therapy in degenerative joint disease, as our present concept is that this is a process of ageing and wear and tear, and that there is no primary inflammatory process within the joints in these individuals. Likewise, gold therapy is of no value in gout, so-called fibrositis, subdeltoid bursitis or specific infectious forms of arthritis.[7] Freyberg and his co-workers have demonstrated that soluble gold enters practically every cell in the body shortly after injection, and that, although a small amount is excreted through the intestines and bronchial mucosa, most of that which is excreted comes out through the kidneys (approximately 80% of the 20% which is excreted). Using a highly accurate and sensitive chemical method,[8] they determined the gold content of blood and the excretion of gold during and after treatment with different gold salts.

In summary, the use of myochrysine or solganol-B in aqueous solution or in oil had comparable effects. In oily suspension, the speed of absorption is reduced. Of the gold injected, about 80% remains in the body for a considerable time. Toxicity usually does not occur until the patient has had several hundred milligrams and, to date, there is no known way of foretelling when or why it takes place. However, the recent work with BAL[9, 10] has yielded most encouraging results. In our series, the several cases in which it seemed indicated have all responded in a satisfactory manner. The employment of BAL, when necessary in the few cases of rheumatoid arthritis which were on maintenance dosage, resulted in relapse of the rheumatoid arthritis and the cure of the gold intoxication, which is an interesting suggestion regarding the therapeutic effect of gold salts, the hypothesis being that the toxic effects in rheumatoid arthritis are produced by the inactivity of the sulfhydryl group in the enzyme proteins in the host, and that the therapeutic effect of gold is due to a similar affinity for and replacing of this group by gold, thus removing or partially displacing the related symptoms. When the action is reversible and the gold salts in their turn produce toxic results, they are similarly affected and removed by BAL. Theoretically, this, of course, offers some explanation to the pathogenesis of rheumatoid arthritis.

In conclusion, therefore, it must be admitted that how gold works in rheumatoid arthritis is not definitely known. Pregnancy also has a temporary alleviating effect, as has jaundice, both without pharmacological explanation. Presumably, therefore, there may be some common factor in the effect of pregnancy, jaundice and the administration of gold salts which, for the time being, might be designated as the "X" factor. This we do know: (1) Gold enters almost every cell in the body shortly after injection. (2) The greatest concentration is in the liver, spleen and kidneys. (3) The plasma gold concentration is between 0.04 and 0.08 mgm. per 100 c.c.

DOSAGE AND METHOD OF ADMINISTRATION OF GOLD SALTS IN RHEUMATOID ARTHRITIS

The use of gold salts in the treatment of rheumatoid arthritis is still in the experimental stage, and requires experience and constant laboratory supervision. Today, it is generally acknowledged as the best available remedy, combined with general measures, for the treatment of this disease. Our objective, however, is to obtain a method of treatment which can be safely utilized by those in general medicine. Unfortunately, the toxicity inherent in chrysotherapy makes it unsuitable for practitioners without special experience, but there are many valuable forms of treatment in medicine which should be administered by those especially trained, and the modern trend seems to be towards co-operative or group division for first-class medical treatment. Be that as it may, it must be admitted that there are many alarums and excursions in the course of gold therapy, and usually therapeutic results are not so satisfactory in the hands of the inexperienced. So far as

the inherent risks of the treatment are concerned, as Philip Hench[11] has pointed out: "Surgeons and patients do not hesitate to accept the risks of cholecystectomy or hysterectomy to relieve symptoms much more bearable than those of progressive rheumatoid arthritis. Yet the mortality rates of such procedures are as great or several times greater than those of chrysotherapy".

In view of all the foregoing, the use of gold salts seems entirely justified (1) in cases of progressive rheumatoid arthritis unrelieved by a reasonable, but not too long, period of older and safer methods of treatment; (2) when the patient clearly understands and accepts the risk; and (3) when the physician is in a position to give the treatments with the necessary clinical and laboratory safeguards.

So far as the actual method of treatment is concerned, there is some slight variation. In Montreal, at the Royal Victoria Hospital we use solganol-B oleosum, and at the Queen Mary Veterans' Hospital, myochrisine. To date, no appreciable difference has been noted in their therapeutic effect. We give all injections intramuscularly. The doses are in milligrams and always given at weekly intervals. The first dose is 10 mgm., the second is 25 mgm., and the third and subsequent ones 50 mgm. until a total of 985 or 1,035 mgm. has been administered. This means twenty-one or twenty-two weeks' treatment. Subsequently, our plan is to continue at monthly intervals with 50 mgm. for sixty months in all, if, in the interval, nothing better is discovered for the treatment of this disease. The reason for setting the period at five years is because, according to the standard of rheumatologists, no case of rheumatoid arthritis is considered as cured until it has remained inactive for five years. Otherwise, it is categorized as an arrested case. The reversibility of the disease has been noted on several occasions, and, in some cases, the period shortened to two or three weeks, and, in others, the amount increased from 50 to 100 mgm. in large men.

We have not treated with gold salts any children suffering from rheumatoid arthritis, but, obviously, in children smaller doses should be used. During the treatment, a complete examination with urinalysis and a hæmogram is made monthly; and weekly, the patient questioned regarding any untoward symptoms, blood pressure is recorded, mouth, throat and skin inspected, and routine urinalysis carried out. If any evidence of toxicity appears, gold therapy is temporarily withheld, and further indicated investigations carried out. Under the above system of dosage, this rarely occurs when the following absolute contraindications to gold therapy have been eliminated: history of purpura, agranulocystosis, renal or hepatic disease, pregnancy, hæmophilia, severe diabetes mellitus, colitis, severe anæmia or any hæmorrhagic tendency, severe eczema or chronic dermatitis, severe bronchial asthma, or any serious systemic disease other than rheumatoid arthritis or psoriasis. The commonest complications which occur following the administration of gold salts are the various dermatitides (one of the chief characteristics being extreme itchiness), stomatitis, presence of albumin or red blood cells in the urine, G.I. symptoms such as nausea and epigastric distress. After a suitable period of observation, the gold injections usually can be continued, but if the symptoms reappear, as a rule injections of gold salts are discontinued. When the following complications occur, gold injections are permanently discontinued: purpura hæmorrhagica, severe exfoliativa dermatitis, ulcerative enteritis, acute yellow atrophy of the liver, aplastic anæmia and agranulocytosis.

A few patients complain of additional pains in the joints and temporary increase in joint swelling and stiffness for twenty-four hours following an injection. Usually after the seventh or eighth injection, the pain lessens and the swelling commences to subside. Later, the sedimentation rate begins to fall. Sometimes improvement does not occur until the end of the period for weekly injections, and, in a small group of cases, the pathological change is irreversible. The most satisfactory results are, as a rule, obtained in the early cases when the inflammatory process is chiefly limited to the soft tissues. In the more advanced cases, with marked muscular atrophy and cartilaginous erosion, the results are, of course, not so spectacular. However, even in these patients, the inflammatory process may be checked and the swelling and pain reduced.

SUMMARY

The present status of therapy in rheumatoid arthritis is reviewed, especially in reference to the use of gold salts, with discussion of methods now being more generally employed and detailed description of those in force at

the Queen Mary Veterans' Hospital and the Royal Victoria Hospital in Montreal.

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RÉSUMÉ

Avec l'usage de fortes doses de vitamine D, celui des sels d'or demeure actuellement au premier plan dans le traitement des arthrites rhumatoïdes. Forestier employa le premier les sels d'or, par la voie veineuse, mais on préfère de nos jours l'injection intra-musculaire. Les sels les plus employés sont le thiomalate d'or et de sodium (Myochrysine) et le thio-glucose aurique (Solganol-B) en suspension huileuse. Chacun de ces sels contient environ 50 p. 100 d'or métal et se donne en doses hebdomadaires de 50 milligrammes, sauf pour la première qui est de 10 milligrammes, et la deuxième de 25. Au bout de vingt-et-une semaines, on espace les injections d'un mois. Les sels d'or permettent l'arrêt du processus arthritique dans un grand nombre de cas, après l'essai infructueux des autres moyens thérapeutiques (physiothérapie, immobilisation, repos général, élimination des foyers infectieux, transfusions). Il importe cependant de faire appel à tous ces moyens avant de recourir à la chrysothérapie, à cause des manifestations toxiques dont celle-ci peut s'accompagner. Elles comprennent une dermatose prurigineuse, une stomatite, l'albuminurie ou l'hématurie, la nausée. Des complications plus graves, purpura, entérite ulcéreuse, atrophie jaune aiguë du foie, anémie aplastique et agranulocytose, contre-indiquent absolument la poursuite du traitement. Il y a avantage à se servir du BAL dans les cas d'intoxication par l'or.

PAUL DE BELLEFEUILLE

EXPERIENCES IN PERIODIC HEALTH WORK*

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WITH the object of trying to give some idea of where the periodic health examination fits into personal preventive medicine, I presume to relate some impressions derived from sixteen years' experience in directing the periodic health work for three of our Canadian Life Insurance Companies (Confederation Life Association, The London Life Insurance Company and the Sun Life Assurance Company of Canada), and also one of our large industrial organizations.

The prevention of disease falls into two parts: (1) That directed by our Public Health Departments, which can be termed "Community Preventive Medicine". (2) That directed to the individual which is "Personal Preventive Medicine" and which is subdivided into "Immunization" and "Periodic Health Examinations". Therefore, it is noted that periodic health examinations are an integral part of preventive medicine, and it is in the use we can make of these examinations that I wish to make some suggestions.

The ordinary men and women we meet every day are more interested in their health than they were twenty years ago. This is evidenced by the increasing number of syndicated health articles in our daily and weekly press and by articles on health (?) in our popular magazines. We, the profession, know that many such articles are not worthy of attention by any thinking man or woman, but do the public know that? An article on cholesterol as the cause of high blood pressure, etc., appeared recently in one of the most widely read of the monthly periodicals. Shortly after its appearance the writer was having lunch with some business men. Three-quarters of them had read the article and some were quite prepared to forego milk and eggs for the rest of their lives on the basis of the article. We know the accuracy of information thus obtained is often in serious question. What are we going to do to correct the situation?

The public are extremely interested in health, and it devolves upon us—the medical profession—to see that their interest is an intelligent interest, and the advice they get is accurate and to the point. The interest could, and too often does, become a morbid one. The annual health examination, conducted by the family physician, is our best means of preventing this.

'Health can be improved, life expectancy and physical efficiency can be stepped up and last, but very important, a more comfortable old age can be assured. There is no use in endeavouring to extend the life span unless the individual is able to enjoy it. A grouchy old age is not desirable. These things can be ac-

* An address to the final year class in Medicine at the University of Western Ontario, London, Ont., November 17, 1947.

complished by the periodic health examination, a fact which is recognized by insurance and industrial organizations. The Canadian Medical Institute has, since its inception, distributed over half a million dollars to the physicians in Canada. This expenditure would not be justified if it was not recognized as good business. In the United States and Canada industry spends millions of dollars every year for medical services. The fundamental procedure of industrial medical services is the annual health examination.

An example of how an industry appreciates such a service is George Weston Limited—a large baking and confectionery organization operated in Canada from Halifax to Victoria. Some years ago they started medical service in one plant. Then it was extended to all their large plants in Canada. All of these plants were given medical services, including annual health survey. This covered 4,800. Finally, in 1946, a periodic health examination was offered to every employee in Canada. This took in employees of smaller bakeries, salesmen, district office staffs, etc., about 1,100 in all. In the Weston organization the medical service shows a profit the same as any assembly line. That is the only basis on which medical service should be carried on in any industry.

The physician should encourage his patients to come to him for an annual health examination. If the periodic health examination works to the benefit of industry and insurance companies, it must be to the advantage of the individual. Its use should be encouraged by all means in the power of our profession. It should not be left to industry and insurance companies to lead in this work. They cannot begin to cover the whole population.

The periodic health examination is intended for the apparently well individual, and the advice given leads to correction of faulty living habits. Such correction helps to prevent the onset of disease. In addition, the periodic health examination with its attendant advice controls incipient disease conditions. An illustration of this is the experience of the Canadian Medical Institute: 1.4% of all examinations done showed glycosuria. A few of these were, doubtless, low thresholds, but a good percentage can be assumed to be incipient diabetes. The same percentage holds good in industry. The majority are early cases and are being controlled by diet. Detecting this condition, before the arterial system is impaired, undoubtedly adds years to the life expectancy. The findings most frequently noted in the annual health examination are bad health habits, overweight, malnutrition from faulty diet, too much or too little exercise, worry, lack of rest and relaxation. These result in functional disturbances, especially affecting the digestive tract, circulatory and nervous systems.

The insurance companies have determined that expectancy of life is lower in those who are overweight and who have increased abdominal measurement. This is proportionate to the amount of excess. For the other findings mentioned there are no specific figures available. We cannot evaluate bad health habits in the form of statistics.

In 1932 the Ontario Medical Association had a committee appointed to study the cause of degenerative diseases. Their final report was submitted in May, 1934, and one of their recommendations was:

"Your Committee further suggests that the study of a group of medical practitioners be engaged in keeping careful records of a large group of young persons over an extended period, so that ultimately the gaps in our knowledge of the rôles played by heredity and infection in degenerative disease be closed up and preventive measures supplied."

What was this but to put into practical use the periodic health examination? That was fifteen years ago but the recommendation was not acted upon.

The experience of the Canadian Medical Institute with one of our most common degenerative diseases (*i.e.*, coronary thrombosis) may be of interest. During the past 17 years we have had over 800 coronary deaths and, reviewing the examination reports, we found the average time intervening between examination and death to be three years. We found that over 75% of these coronary fatalities had, at the time of examination, one or more of the following impairments: overweight, increased abdominal measurement or hypertension. How much of this could have been prevented if the individual had had yearly contact with his own physician? In a previous paper, we showed that hypertension could be helped and controlled by reduction of weight.

Dr. W. R. P. Emerson, author of numerous articles and books on physical fitness, is well known for his work as medical consultant on physical fitness for the Aetna Life Insurance Company, in a personal communication to the writer, he stated that he is not so concerned with the reduction of weight as he is with the reduction of food intake, which results in relief to an overburdened metabolism. In his experi-

ence, relief of the latter definitely did reduce hypertension.

Who should give health advice but the man's own family doctor? The family physician can get nearer to his patient than anyone else. What more intimate personal relationship can be established than through a friendly talk devoted to telling a man how to keep well? The same can be said for the competent medical officer in industry. Here the author can say that it definitely pays to take time to talk to employees and advise them how to remain fit. Advise him about his overweight, bad habits, etc. Our physical stature is complete at 18 years of age, and then at about 40 we start growing at the middle! The physician can advise his patient about the handicap he will develop if the tendency to overweight is not corrected. Here I should like to quote from Dr. Charles Hunter's article on "Minor Ailments" in the February issue of this *Journal*:

"Overweight—a forgotten disability.—We physicians have a very bad record in dealing with overweight in our patients. We do not take the condition seriously. We shut our eyes to the long range dangers involved and so do not arouse and stiffen the patient's fight against obesity. In these days of preventive medicine, we neglect to warn younger members of stout or diabetic families; through our indifference, too often even pronounced cases turn to the quack, the beauty specialist or the dietary crank for advice on this subject. Yet the evils of overweight are well known and undisputed. The mortality figures of every insurance company emphasize the shortening of human life involved and might profitably be studied by the sceptical physician and patient alike."

Recently a panel discussion on health in industrial executives of the General Motors Corporation was reported by Dr. Edwin DeJongh in the *Journal of Industrial Medicine*. This discussion brought out that the most important finding among executives was obesity. As high as 23% were suffering from this disability. During the discussion, Dr. R. L. Haden stated that "in his opinion cardiovascular disease was the fundamental problem. About 25% of the executives examined were affected. Impressive, of course, was the number of associated obesities. He thought it more important to help a man change his habits and lose some weight than to worry about cryptitis."

Advice should also be given about proper nutrition—eating a balanced diet. A person doesn't need vitamin pills except on his physician's prescription *if* he will eat properly. In this regard, let me quote from an article by the late Commissioner of Health for Michigan, Dr. S. M. DeKline:

"I venture the opinion that, when and if we learn to apply the knowledge we now have about nutrition, deaths from many of the degenerative diseases (including heart) will go down. The literature which has accumulated on the subject of nutrition clearly indicates a very close relation of food to health and disease. . . . Carry with you the conviction that the science of nutrition is of paramount importance. It holds the key, I believe, to the cause of many forms of illness."

There is no argument about improper diet. We all know the benefits derived from correction, but why should the patient have to get his information from newspapers, magazines and quacks? It should come from his family physician.

Faulty health habits can be corrected. We do not know exactly what bearing they have on disability, but we believe that high tension living without periods of rest and relaxation, worry, etc., are factors in causing functional and possibly organic disturbance.

What can we do about this? The average intelligent man can correct part—if not all—of his habit of worrying. The worry habit cannot be stopped without replacing this mental activity with something new. How about the man who lives at high tension? This man has a day full of responsibilities. He is always working against time. He gets up at 7 a.m., rushes through breakfast, and rushes to the office where he works at full speed until lunch time. He probably snatches half an hour for lunch and then goes back to the office where he works until late in the afternoon. Six o'clock sees him home in time to read the paper. Dinner is a little later and it, too, is probably rushed if he has business meetings or social engagements. This is a very long day. A rest between five and six would be beneficial and would be taken if the necessity for it were explained. Who can best explain this? The family physician!

A case history from the records of the Canadian Medical Institute will illustrate my point.

A physician aged 45, with a successful practice, working very hard. At periodic health examinations for the previous five years the blood pressure had averaged 120/76. The sixth examination showed a blood pressure of 146/92. He was advised by his examiner to slow up in general and go to bed from five to six every afternoon in particular. Six months after following this advice regarding a rest period, his blood pressure was 140/90. In one year the blood pressure had returned to normal, and today at the age of 57, it is 118/76. This man still works hard but *always* takes his daily rest.

Since 1931, when the Canadian Medical Institute started offering periodic health examinations to policyholders of our companies, there has been a very marked improvement in the advice given by the examining physician. For the benefit of those of the profession who do not do these examinations, we should like to state that our policy has always been for the examiner to give the advice to the examinee. We feel that the examiner—in contact with the policyholder—can give much better advice than that coming from a central office. We, in our turn, back up the advice of the examiner, giving emphasis to his advice when needed. In addition, general health advice is given where it may be required. Fifteen years ago, the examinations were, in many cases, conducted like the examination a man has before taking out life insurance. The doctor was obviously looking for organic disease, gross errors in living conditions, etc., rather than tracking down and correcting tendencies which would make organic disease a certainty in the future. Today that is to a great extent changed for the better. The advice now is much more on health and the maintenance of a healthy body than formerly. To a great extent this may be due to the teaching in our universities. During the last ten years every medical school in Canada has given at least one hour's lecture on the value of the periodic health examination. This is a definite step in the right direction. High blood pressure and organic heart impairment may be definitely established before the patient is warned by symptoms. In most cases these symptoms are discovered at the routine health examination, and something can be done before it is too late. It is not such a shock to learn that you may develop disease as to learn that you have it.

However, there is still room for improvement in the periodic health examination, particularly in the method of giving advice and in the information given to the examinee. Bear in mind that the periodic health examination is for the apparently well person who wants to maintain his health. It is not a periodic health examination when a man, who has obviously been "unwell" for some time, comes to your office and asks for a check-up. Bearing that point in mind, let me state that no person should leave the physician's office after a periodic health examination with fear in his mind. If he is pronounced fit, he should be grateful. If any impairment has been found he should also be grateful, although it is only human that he wouldn't be so happy about the latter finding. This is again the time for the family physician to step into the rôle of adviser and friend.

One of the most common fears the laity have today is the fear of high blood pressure. This need not be so. Now, what are you going to tell a case of elevated blood pressure? The most important thing to be considered is what not to tell him. First and foremost, except in unusual circumstances, no case should be told the blood pressure readings. I was interested to hear Dr. Wright Young, Bell Telephone Company, Toronto, in an address in Montreal last October, state that they did not tell people their blood pressure reading. If more physicians followed this plan it would be better for the public. In the reports that go out from our office no person with hypertension is ever told the examiner's blood pressure findings. The other extreme of the blood pressure reading—*i.e.*, hypotension—is too often suggested to the examinee as being an impairment. Low blood pressure is usually not significant where there is no organic or infectious disease present, where the individual is free from overfatigue, infection of chest, teeth, tonsils, etc., and is taking sufficient exercise. Quite frequently these people, who have, obviously, no other impairment, are cautioned against exercise, advised to rest and to take medication.

In doing a periodic health examination keep in mind that faulty health habits, unbalanced diet, poor chest expansion, overweight even as little as ten pounds, lack of rest and relaxation, may be the cause of physical disability in the future of the examinee.

In conclusion, let me again urge you to give health advice to your patients by means of periodic health examinations. No one can do it better. It will be appreciated and, if practised by all, will result in a more satisfied and a healthier public. I cannot think of a better time to thank the two thousand or more physicians in Canada who do examinations for us every year. I wish to commend them for the care and attention they are giving our examinations.

Room 606, 302 Bay St.

CASE REPORTS

COMPOUND DISLOCATION OF ELBOW JOINT WITHOUT FRACTURE

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The following case of compound dislocation of the elbow joint without fracture is being reported because of some of the interesting features which it exhibits and because reports of these cases are very rare. It is very common to have fracture dislocations of the elbow but actual compound dislocations unaccompanied by fracture are few and far between. A few such cases have been reported, one a late case reported because of orthopædic treatment some years after injury and one which was associated with rupture of the brachial vessels. Our case was quite uncomplicated and has made a full recovery. It is detailed below.

The patient, a stout middle-aged woman suffered an injury on September 14, 1946. She fell down some cement steps against an iron railing crushing her elbow and twisting her forearm. This caused a compound dislocation in which the humerus projected through the skin. She was attended by Dr. H. O. Singer of Welland and brought to the Welland County General Hospital. X-ray examination was entirely negative for fracture but a lateral dislocation of the forearm was present.

Clinical examination showed the humerus projecting through the skin at the medial side of the elbow joint from a 3″ laceration. There was slight duskiness of the hand and considerable swelling about the elbow. The radial pulse though not as good as on the opposite side was fairly satisfactory. She also had suffered a 6″ laceration in the right leg below the knee which is unimportant. Her general condition was very good so she was taken to the operating room soon after admission. A great deal of time was spent preparing the skin and finally the laceration was lengthened and debrided. The triceps tendon and joint capsule were frayed and this damaged tissue was excised. The joint was inspected and found intact except for the dislocation. This could readily be reduced but recurred until the rent in the capsule and the triceps tendon had been repaired with chromic catgut. The ulnar nerve was found lying near the rent in the capsule and was apparently uninjured. We were afraid that it might be included in scar and for this reason it was dissected free both upwards and downwards and transplanted under deep fascia at the front and the medial side of the elbow joint in the upper part of the forearm. At the end of operation the elbow joint appeared to be perfectly reduced and it had full motion and was stable. It was placed in a padded cast at a right angle. The wrist was in mid-position. The skin of the incision was not closed. Penicillin 100,000 units was injected into the joint. She was given penicillin for 4 days postoperatively as well as sulfathiazole by mouth.

On September 25, 1946, the cast was removed. Secondary suture of the incision was done but this could not be completed until a postero-lateral relaxing incision was made. This bare area was immediately covered by a split-thickness skin graft from the abdominal wall. A right-angled cast from axilla to palm was again applied.

COMMENT

This case clearly demonstrates the value of chemotherapy and early operation. The good result achieved could not have been possible without the use of advances such as these in treatment.

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BACTERIAL ENDOCARDITIS (STREPTOCOCCUS VIRIDANS) IN AN EIGHTEEN-MONTHS OLD INFANT SUCCESSFULLY TREATED WITH PENICILLIN*

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Bacterial endocarditis has been described in all decades ranging from birth[1] to extreme old age. It is, however, relatively rare in infancy, where the underlying basis is almost invariably a congenital cardiac defect, most commonly a patent interventricular septum or patent ductus arteriosus.[3] Sansby and Larson[2] reviewed the world's literature in 1930 and found only 5 cases of acute bacterial endocarditis in children under 7 months of age.[2] Gelfman and Levine,[3] reporting 34,023 autopsies, noted only 5 cases of congenital heart disease with bacterial endocarditis below the age of 2 years. Davis and Weiss[4] reviewed 5,125 consecutive autopsies and recorded no cases of the disease under the age of 10 years. In 5,250 reported cases of bacterial endocarditis of Kelson *et al.* the youngest patient was 2½ years old. Christie's[6] recent account of 269 cases of endocarditis treated with penicillin indicates the youngest to be 7 years. A search of the literature up to May, 1948, appears to show only one recorded case of endocarditis, the child not surviving.[7] For this reason, it has been thought worthwhile to report the following case of *S. viridans* endocarditis, in an eighteen-months old baby.

D.E., an eighteen-months old white male infant, was admitted to the Royal Victoria Hospital on December 18, 1947. Both parents were healthy and this was the first child. The baby was full term, weighed 6 lb. 3 oz. at birth and labour was said to have been easy.

* From the Department of Pædiatrics, Royal Victoria Hospital, Montreal.

The parents were told shortly after birth that the infant suffered from a cardiac abnormality. There was no history of rubella or any illness in the first trimester of the mother's pregnancy.

From birth to 5 weeks before admission the child seemed remarkably healthy. It was raised on an exemplary pædiatric regimen, received the usual inoculations and was never observed to be ill in any way. The parents noticed no dyspnœa or cyanosis. It developed at a normal rate, weighed 23 lb. at one year and appeared active, bright and happy.

Five weeks before admission, the child became cranky, lost its appetite and seemed feverish. A physician treated the baby over a period of one month with sulfonamides but the child developed increasing irritability, pallor, and listlessness, lost much weight and was persistently febrile. The parents, one week before admission, noticed the child seemed dyspnœic and blue when crying. Further inquiry contributed nothing to the history, except that the baby had scratched himself incessantly for the past 2 weeks.

Physical examination revealed an emaciated infant with a ghost-like pallor. He seemed exhausted and seriously ill and in almost a frenzy of irritability and fear. There was marked dyspnœa and the lips showed a faint lavender cyanosis. Ears, mouth, nose and throat were not remarkable except for the pallor of the buccal mucous membranes. There was a tiny petæchial hæmorrhage in the left lower conjunctiva. There was minimal generalized lymphadenopathy. The respiratory rate was 50 per minute and the child was frankly dyspnœic. The lungs were clear. A slight systolic thrill was felt over the præcordium. A loud, harsh, short systolic murmur obliterated the first sound over the præcordium and was best heard over the third interspace to the left of the sternum. The apical rate was 170 per minute and regular. There was no venous engorgement. The liver was firm and palpable 3 fingers' breadth. The spleen was just palpable. Multiple fresh and old purpuric spots were seen over the neck and forehead. Almost the whole body was covered with scratch marks. There was no œdema. The rest of the physical examination was not remarkable. There was no jaundice.

A diagnosis of patent interventricular septum with a superimposed bacterial endocarditis was made. The child was seen by Dr. G. R. Brow, who agreed with the diagnosis of interventricular septal defect.

Investigation showed the red blood cells to number 2,800,000 and white blood cells 28,000 with a differential of 21 stabs, 57 polymorphonuclears, 18 lymphocytes and 8 monocytes. The hæmoglobin was 10.1 gm. %. The sedimentation rate was 9 mm. per hour. The platelets numbered 160,000. The coagulogram was within normal range. The Wassermann was negative and old tuberculin (1:1,000) negative, as was the urine. An electrocardiogram on December 19 showed an A-V conduction time of 0.1 seconds. The rate was regular and 170 per minute. There were moderately deep Q_1 and Q_2 deflections and T_4 was diphasic. This was repeated on February 9. It was essentially the same except for the appearance of a Q_3 spike and T_4 had reverted to a positive deflection. Non-protein nitrogen measured 22.1 mgm. %, total protein 6.37 gm. %, with albumen 3.56 gm. %, and globulin 2.81 gm. %. Cephalin cholesterol was 3 plus, thymol turbidity 9.7 units, and thymol flocculation 5 plus. Chest film showed the heart to be moderately enlarged and globular in shape. The broncho-vascular patterns were intensified. Blood cultures on December 21 and 22 grew 40 colonies of *S. viridans* per ml. of blood and 200 colonies per plate respectively. The bacteria were inhibited by 0.05 units of penicillin.

On admission the child was transfused with 250 ml. of whole blood.

Report of the positive blood culture was received on December 22 and on this date the infant was empirically placed on 125,000 units of penicillin given every three hours, as it appeared too critically ill to consider waiting for the report on the penicillin sensitivity of the organism. This dosage gave a level of one unit of penicillin per ml. of blood at the end of 3 hours. The organism was found to be sensitive to 0.05 units of penicillin. Despite this overwhelming dosage the child continued febrile, up to 102° F. (rectal) daily, for a further period of 8 days, and the white blood cells remained around 30,000. By January 1, after ten days of treatment, the temperature became essentially normal and on January 4 the penicillin was reduced to 60,000 units every three hours, giving a penicillin level at the end of 3 hours of 0.5 units per ml. Over the next 2 weeks the temperature fluctuated between 99.6 and 101° F. (rectal) and the white blood cells fell to 14,000. Clinically, the infant did not seem much improved. In this period the baby was given one gram of caronamide (4-carboxyphenylmethane sulfanilamide) every 4 hours. This did not raise the penicillin level. It was increased to 2 gm. every 4 hours, but this specimen of blood for penicillin level was unfortunately broken.

On January 17 (25 days after onset of treatment) the child became more dyspnœic and cyanosed and the temperature rose to 104.5° F. (rectal). A friction rub was detected in the right chest and signs of pleurisy and effusion in the area developed over a period of 2 days which were confirmed by roentgenogram. The white blood count rose to 36,000.

At first it was considered that this might represent an embolic phenomenon, but as the fever and signs persisted it was felt that the infant had developed pneumonia and pleurisy from a penicillin-resistant organism. Actually, naso-pharyngeal swabs grew no pathogens. The child was given sulfadiazine (one gr. per lb. per day) and within 72 hours the temperature fell to normal and remained so till discharge. Co-incidentally, there was marked clinical improvement. The dyspnœa decreased and the appetite became almost voracious. The infant, who had been listless, irritable and mute, uncovered a bright, active, talkative nature and gained weight. In the meantime, the spleen had disappeared and the liver measured only one finger breadth below the costal margin. The skin ceased being irritable and the scratching stopped. Six blood cultures, at weekly intervals, were sterile. Penicillin had been given for 39 days and totalled 26,000,000 units. The baby was discharged on February 16 looking much improved. The heart murmur was much the same. At this time the hæmoglobin was 12.6 gm. %, and white blood count 7,200. Interestingly, three months later the red blood count numbered 5,700,000 and the hæmoglobin 17.6 gm. %.

The child has continued to do well and gain weight, but shows a degree of fatigue and dyspnœa on exertion which was not present before his illness. This would suggest some further damage to the heart from the bacterial invasion. One might suspect an increase in the size of the septal defect and some mixing of arterial and venous blood in view of the relatively marked erythrocytosis which has developed since his discharge from hospital. Six months following the termination of treatment, the child has shown no signs of relapse.

SUMMARY

A case of *S. viridans* bacterial endocarditis in infancy has been reported. The child was treated with massive doses of penicillin with apparent complete recovery.

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BENIGN STRICTURE OF THE LOWER ŒSOPHAGUS TREATED BY RESECTION AND ŒSOPHAGO-GASTRIC ANASTOMOSIS

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Mr. D.W.K., aged 39, was admitted to Westminster Hospital December 27, 1945, complaining of (1) upper abdominal pain—4 weeks' duration; (2) vomiting—4 weeks' duration.

He had been discharged from Westminster Hospital on November 8, following treatment for bleeding "peptic" ulcer and had returned to work for one week when he had a recurrence of the above symptoms. He remained at home one week on a milk diet and then was advised to enter a hospital. The vomiting and pain became worse. There was some blood in the vomitus.

Past history.—He developed "indigestion" in 1941, which was relieved by "stomach powder". In March, 1945, he had a perforation of a duodenal ulcer which was repaired. He was in hospital five weeks and returned to duty at the end of three months. He was discharged from the Army, Category E, in May, 1945. He remained well until September, 1945, when the pain recurred and he had a severe hæmorrhage for which he was admitted to Westminster Hospital.

The functional inquiry was negative and the family history contained nothing relevant.

Physical examination.—He was thin and pale. Blood pressure 112/84. Examination was negative except for the abdomen which was scaphoid, with notable pulsation in epigastrium, synchronous with pulse. Upper right rectus scar present, well healed. Marked epigastric tenderness on palpation but no rigidity. No visible peristalsis.

Urinalysis, acid, 1.012 to 1.032 specific gravity. No albumen or sugar. Hæmoglobin, 70%; red blood cells, 4,200,000; white blood cells, 7,300; standard and presumptive Kahn, negative. Fæces negative for occult blood; non-protein nitrogen, 30 mgm. %; sedimentation rate, 6 mm.; fasting gastric analysis in May, 1945, had shown free HCl. 70. Barium swallow and meal on September 21, at Westminster Hospital showed a normal œsophagus and stomach except for evidence of increased gastric peristalsis. The duodenal bulb seemed fairly well formed on the screen but subsequent film studies all showed an old cicatricial distortion, without evidence of active ulcer crater or obstruction.

Gastro-intestinal series which had been carried out at another hospital on December 15, were reported as showing a gastric ulcer high on the lesser curvature of the stomach close to the œsophageal entrance, and also a duodenal ulcer which caused considerable delay in emptying of the stomach. Barium meal on December 28, showed considerable change in the upper G.I. tract since the previous examination. Some delay was noted at the lower end of the œsophagus which appeared narrowed. A definite sharp filling defect about three-quarters of an inch in length was present above the cardiac orifice. Two subsequent barium meals with radiological studies on January 28 and February 13, 1946, respectively, failed to show evidence of œsophageal obstruction. A small duodenal ulcer without obstruction was reported.

The patient was placed on medical management following a surgical consultation. He continued satisfactorily until March 1, when he began to have difficulty in swallowing and lost about 10 pounds in weight. Another barium swallow was carried out on March 11, which showed "a rather abrupt stoppage to the barium flow at a point one inch above the cardiac orifice. From this point a tiny trickle of barium flowed into the stomach". Œsophagoscopy was done on March 28, by Dr. F. S. Kennedy. The whole length of the œsophagus was observed. There was no evidence of stricture down to the cardiac orifice. It was suggested that the patient was suffering from early cardiospasm. The patient was given a thread to swallow and dilatation by means of bougies was attempted. A 35F dilator was completely blocked and no further attempt at dilatation

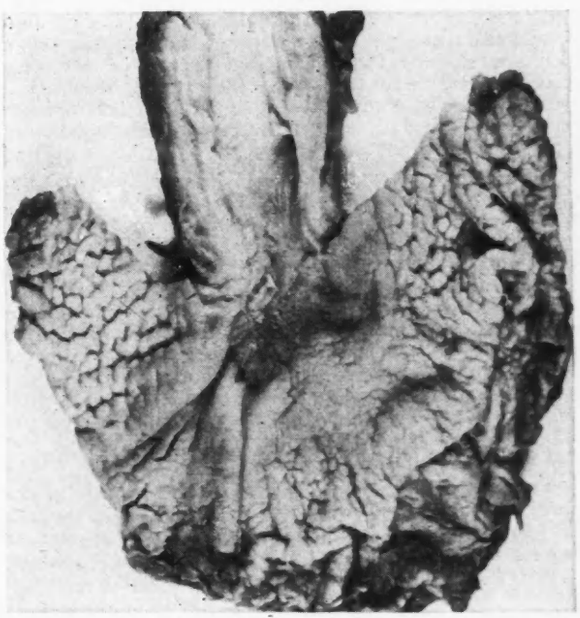

Fig. 1.—Microscopic examination revealed diffuse inflammatory infiltration of the thickened portion of the œsophagus with lymphoid hyperplasia of the associated lymph glands.

was carried out. However, there was some improvement in his swallowing, but obstruction to solid foods persisted.

Œsophagoscopy under general anæsthesia was again carried out on June 14. The œsophagus was observed down to the cardiac orifice but the œsophagoscope did not pass the cardia. Attempted dilatation of the stricture at the cardia was unsuccessful. It was felt that there was an organic stricture of the œsophagus in the cardiac area that might be malignant.

The patient was transferred to the Surgical Service after surgical consultation and on July 9, Dr. A. J. Grace carried out a one-stage partial œsophago-gastrectomy. The œsophagus was found to be bound down by dense fibrotic tissue which was gradually freed by means of blunt and sharp dissection. The left phrenic nerve was blocked with novocaine. The diaphragm was incised and the stomach and lesser omentum explored. Several small firm glands suggestive of malignancy were palpated in the lesser omentum.

Approximately one and one-half inches of the cardiac end of the stomach was resected together with a large

part of the lesser omentum following division of the left gastric artery and vein between ligatures. The distal portion of the stomach was closed with clips (Von Petz clamp), continuous intestinal chromic catgut, and a layer of interrupted No. 50 cotton sutures. The œsophagus was then freed by blunt dissection up to behind the left lung root. Section of the œsophagus one-half inch below the left lung root was carried out between intestinal clamps and the specimen was removed.

The stomach was then drawn up into the left pleural cavity and anchored by means of No. 50 cotton sutures to the posterior and mediastinal parietal pleura well above the lower end of the remaining portion of œsophagus, to prevent any tension on the anastomotic suture line. A three layer anastomosis between the œsophagus and stomach was then carried out by interrupted No. 50 cotton sutures according to the technique of Sweet. The diaphragm was repaired and the left phrenic nerve crushed. The wound was closed in layers, a catheter being left in the left pleural space through a stab wound in the ninth intercostal space for closed drainage and controlled suction.

Pathological examination of the specimen showed the œsophageal portion to be considerably thickened, measuring up to 0.8 cm. in thickness. A probe could be passed through the narrowed lumen; the open specimen showed the mucous membrane of the œsophagus to be intact but considerably congested. Immediately distal to the cardiac opening of the stomach there was a small, superficial, ulceration measuring 1 x 0.6 cm.

Postoperative management.—A Levine tube was inserted into the stomach following the operation and Wangensteen drainage was continued for two days, after which feeding through the tube was commenced and continued for fourteen days. Patient was put on soft diet of multiple small feedings. His convalescence was uneventful except for some gaping of the skin wound when the sutures were removed on the seventh day and resuturing was required. He was allowed to leave hospital as an outpatient 22 days after operation and was discharged finally, on November 28, 1946. At that time he had no distress on taking foods. He followed a fairly rigid bland diet and routine with multiple small meals.

This patient has been seen from time to time in the Surgical Clinic, and was last examined on February 11, 1948, when he stated that he was still unable to eat big meals and had gas after food. He was gaining in strength and was not having a great deal of stomach distress unless he got too tired. Gastric analysis at this examination showed complete absence of free HCl. The G.I. series showed: (1) Satisfactory postoperative œsophagogastric anastomosis. (2) Chronic hypertrophic gastritis. (3) Chronic duodenal ulcer with extensive scarring.

DISCUSSION

Œsophagitis occurs more frequently than does any other pathological condition of the œsophagus. Only a small percentage give rise to symptoms (10%). Acute ulcerative œsophagitis is a common finding at necropsy. Reflux of gastric secretions which are irritating to the œsophageal mucosa, is considered the most important etiological factor. It therefore occurs in diseases with vomiting and high gastric acidity. Prolonged intubation is not thought to be an important factor unless pre-existing œsophagitis be present. Œsophagitis is most often encountered in patients who have gall bladder disease, duodenal or gastric ulcer, obstructing lesions of the upper gastro-intestinal tract and œsophageal hiatus hernia.

In most cases the inflammation subsides without leaving any ill effects. However, repeated episodes of œsophagitis may lead to a progressive cicatrization of the lower part of the œsophagus, as occurred in this case. Harrington believes that repeated episodes of ulcerative œsophagitis and healing are usually responsible for the short œsophagus type of diaphragmatic hiatus hernia. Recognition of the fact that œsophagitis is a common complication of disease of the upper part of abdomen is important. Neutralization of gastric secretions with antacids, elevation of the head of the patient's bed, and early corrective surgery are important prophylactic measures. Nasal feeding tubes should be used with some discretion if œsophagitis is suspected.

If cicatrization and stricture occur, œsophagoscopy should be carried out to rule out malignancy. Most benign strictures respond to dilatation over a previously swallowed thread. Only cases not responding to this form of therapy require surgical intervention.

I wish to thank Dr. A. J. Grace, Associate Professor of Surgery, University of Western Ontario, and Dr. C. C. Ross, Director of Surgery, Westminster Hospital, for permission to publish this case.

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RUPTURE OF AORTIC ANEURYSM INTO THE SUPERIOR VENA CAVA WITH OBSTRUCTION*

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The explosive onset of superior vena caval obstruction is dramatic and occurs characteristically when an aortic aneurysm ruptures into the superior vena cava. The following report of such a case is presented because of its clinical interest and to demonstrate how the application of venous catheterization in the investigation established an accurate diagnosis. Another case not previously reported is described briefly, and

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one in which an aneurysm of the aorta ruptured into the auricle of the right atrium is mentioned.

An arteriovenous fistula between the aorta and superior vena cava is generally recognized to be rare. Armstrong, Coggin and Hendrickson[1] in 1939 added 2 cases to 98 previously reported and collected by them from the literature. According to these authors Beevor,[2] in 1832, was the first to report such a case. Seven others were added and are given by reference in a paper on the superior vena caval syndrome by Hussey, Katz and Yater[3] in 1946; they included an additional case of this complication as a cause of superior vena caval obstruction.

Mrs. A., a housewife, aged 44 years, believed she was in good health prior to her present illness. About five o'clock in the afternoon of September 8, 1947, during the act of bending forward to pick up an article, she experienced a snap low in her neck which she stated felt as if something had broken; this was followed rapidly by a sensation of fullness and tightness in her head and neck. Half an hour later a physician observed the face, neck, upper thorax and both upper extremities to be cyanosed. He found the patient frightened and apprehensive but without pain or respiratory distress. A loud, continuous murmur over the upper sternum was reported on auscultation. On admission to hospital, some three hours later, she complained of a choking sensation in her throat and of swelling and increased fullness in her head, neck, face and arms. There was no pain and her breathing was not difficult.

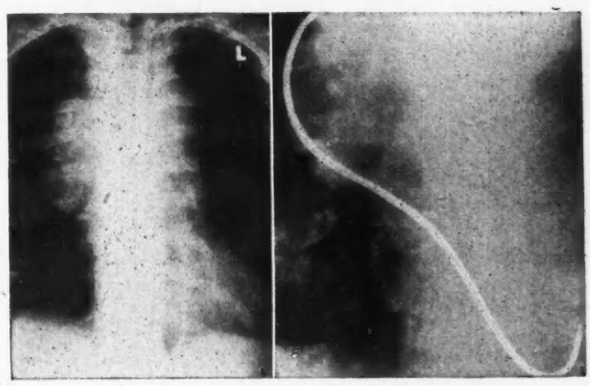

Fig. 1.—Chest film, in the recumbent position, taken immediately after removal of the cardiac catheter. The aneurysmal sac is faintly outlined to the right. **Fig. 2.**—Cardiac catheter, with tip in the right ventricle. Photograph retouched to aid contrast.

Her family history and functional inquiry contained nothing relevant to the present illness. The patient was born and lived all her life in Canada. She had one daughter, aged twenty years and in good health. There had been no miscarriage and there was no history suggestive of venereal disease.

On physical examination the patient was in obvious discomfort, propped up in bed, but not short of breath. There was a deep reddish-blue suffusion in the skin of her face and neck, in both upper extremities and the upper thorax extending down to the level of the second rib anteriorly and the seventh thoracic spine posteriorly. There were numerous superficial venules visible over the thorax, especially at the level of the diaphragmatic attachment to the rib cage. No pulsation was apparent in these venules but peripheral capillary pulsation was present in the nail beds of her fingers. Swelling of the tissues of the neck prevented identification of the external jugular veins. Her finger-nail beds were pink; there was no clubbing.

On examination of the cardiovascular system there was increased dullness on percussion at the level of the second intercostal space; the left border of dulness was estimated to be 4 cm. and the right border 6 cm. from the midline at this level. The apex impulse was palpable 10 cm. to the left of the midline in the fifth intercostal space. Cardiac rhythm was regular at a rate of 90 per minute. The heart sounds were normal at the apex. A loud, continuous, machinery-like murmur with systolic accentuation was heard over the upper part of the chest anteriorly, maximal in intensity over the second right intercostal space, 3 to 4 cm. from the midline. Over this area a systolic thrill was faintly palpable. Widespread transmission of the systolic murmur occurred throughout the right side of the thorax. The diastolic element of the murmur was easily heard along the left sternal border, and was transmitted towards the apex. Blood-pressure in both arms was 98 mm. Hg. systolic, and 45 mm. Hg. diastolic. Both femoral arteries were easily palpable.

The remainder of the physical examination revealed no abnormal findings. The trachea was in the midline and no tug was apparent. The liver was not enlarged. Oral temperature was 100° F.; pulse rate 90 per minute; respirations 24 per minute. Urinalysis and blood count were normal. Serological tests for syphilis revealed 4 plus Kahn and a positive Eagles' test. A repeat Wassermann was requested but not completed. Venous pressure, by direct measurement in the right antecubital vein, was 47 cm. saline. An electrocardiogram was normal. Fluoroscopy of the chest revealed an increase in the area of superior mediastinal density, convex to the right, bulging to a distance of 7 cm. from the midline in the second and third right intercostal spaces. Expansile pulsation was visible in this border. The heart appeared to be pushed downwards and to the left. The superior mediastinal mass appeared to be continuous with the upper part of the cardiac silhouette when viewed in the left anterior oblique position. These findings were confirmed later by roentgen films (see Fig. 1).

The diagnosis made on the first day of admission was superior vena caval obstruction due to aneurysm of the ascending aorta. To explain the murmur, rupture of the aneurysm into the superior vena cava was considered. Positive serological tests for syphilis indicated the

TABLE I.

CASE NO. 18718 (T.G.H.): CARDIAC CATHETERIZATION, SEPTEMBER 11, 1947.

Location of sample	*Mean pressure* *Cm. H_2O.*	*Mm. Hg.*	*Oxygen content volumes per cent*	*Saturation per cent*
Right ventricle	22.0	16	13.6	78.5
Right atrium	4.5	3	14.5	84.0
Superior vena cava	44.0	32	15.9	92.0
Sup. vena cava: 1.5 cm. proximal to previous sample	74.0	54		
Axillary vein	40.0	29	10.5	61.0
Femoral artery			14.8	85.5
Femoral artery recheck			14.5	84.0

etiology of the aneurysm. On September 11, a cardiac catheter passed easily into the right ventricle from the left antecubital vein (see Fig. 2). Manipulation of the catheter was deliberately kept to a minimum. Mean pressures, as shown in Table I, were recorded on a simple manometer of 1 millimetre bore. The procedure was executed uneventfully. Unfortunately her oxygen consumption was not measured.

The high oxygen saturation in the superior vena cava (92%) indicated an arteriovenous shunt of considerable magnitude; delivery of most of this blood to the right atrium (saturation 84%) and further dilution with venous blood in the right ventricle (saturation 78%) was apparent. The oxygen saturation of 85% in the femoral artery—the value was rechecked with another arterial puncture when the first value was known—indicates that some venous blood was drawn into the aorta. This mixing of arterial and venous streams in both directions was an interesting and unexpected finding. A comparison of the mean pressures in the superior vena cava with the blood-pressures obtained on the same day (110/45) with a cuff on the arm strongly suggested that mixing in both directions could occur on dynamic factors alone. The low pressure in the right atrium was a surprise. Marked narrowing of the superior vena caval channel, due to angulation around the lower end of a saccular aneurysm later to be observed, may have been in part responsible.

The diagnosis following this investigation was arteriovenous fistula between the aorta and superior vena cava, 2 to 3 centimetres above the right atrium, due to rupture of a luetic aneurysm of the ascending aorta with superior vena caval obstruction. Thus a fistula was confirmed and its location identified beyond any doubt. The possibility of surgical repair of this lesion was then discussed and rejected.

The patient survived the arteriovenous fistula seven days. During the first and second days in hospital she was subjectively improved: sedation with morphine (1/6 grain, repeated as necessary) and rest in the upright position were employed. The pulse rate rose gradually to 120 per minute during this time, reflecting the increased work of the heart. There was little change in her clinical state during the next three days. On the sixth hospital day she regurgitated three ounces of bright red blood mixed with a few clots; the patient stated that she was certain she had not coughed up the blood. Large amounts of coffee-ground material were regurgitated later in the same evening, and the patient became very restless. Next day her breathing was irregular, her blood-pressure had fallen to 65/30, and her heart rate was 100 per minute. She became comatose and died within a few hours. Death was attributed to cerebral anoxia and hæmorrhage into the gastrointestinal tract; perforation of an œsophageal varix was suggested to explain the hæmatemesis.

Fig. 3.—Saccular aneurysm of ascending aorta; arch is to the right. The dark round shadow in the depth of the sac is a small secondary aneurysm. The perforation is in the lower portion of the sac and is not seen in this view. **Fig. 4.**—Perforation of aneurysm of aorta into superior vena cava into the right atrium below. A white arrow indicates the bifurcation of the trachea. The narrowing and angulation of the superior vena cava below the perforation can be seen.

An autopsy* was performed twelve hours after death. The relevant findings pertain to the heart, great vessels and œsophagus. The heart itself was normal in size and appeared to be displaced downwards and to the left by the aneurysm above it. The muscle, coronary arteries and valves were normal. The aorta was diffusely dilated in the ascending portion and the arch, and it revealed the gross markings characteristic of syphilitis aortitis. A large saccular aneurysm with a circular mouth 6 cm. in diameter projected to the right from the ascending aorta (see Fig. 3). The superior vena cava was compressed by the aneurysm, from the postero-medial aspect, into a crescentic shape; in the angles of the crescent there were fine intimal adhesions. A perforation measuring 6 mm. by 3 mm. in the lateral wall of the sac opened into the superior vena cava 2 cm. above the right atrium (see Fig. 4). The venous channel, in addition to being compressed, turned rather abruptly around the lower margin of the aneurysmal sac proximal to the level of the perforation. These indications of obstruction of longer standing than the present illness were confirmed by the presence of extensive engorgement in the azygos system. There were many small and medium-sized veins visible at the lower end of the œsophagus. A small perforation was present in one of them 3 cm. above the cardia. There was coffee-ground material in the stomach and small intestine.

The hospital records over a twelve-year period, July, 1935 to July, 1947, revealed only 37 acceptable cases of aneurysm of the aorta due to syphilis (31 males and 6 females) prior to the present case. Twenty-three are known to be dead; autopsy reports are available on 14 of them. There was clinical evidence of superior vena caval obstruction in 5 of the 37 cases: two of these are of especial interest.

In one, complete superior vena caval obstruction was recently superimposed on partial obstruction known to be present for approximately four years. This occurred in a woman aged 51 years, who had received radiation therapy in 1944 to an upper mediastinal mass without benefit. She showed a negative Wassermann test at that time. In April, 1947, she became suddenly distressed with a suffocating sensation in her neck without pain, and was intensely blue in her neck and face. The picture of complete superior vena caval obstruction involving the upper extremities and upper thorax was manifest within a few hours: clinically, her heart was normal in size; upper mediastinal dullness was increased, and a to-and-fro murmur was heard over the upper sternum; her Wassermann reaction was later reported strongly positive, but she died before this information was available. An autopsy revealed a large saccular aneurysm involving the ascending aorta and transverse portion of the arch. It had perforated into the superior vena cava through a small opening approximately 4 centimetres above the right atrium. The aorta showed the characteristic lesions of luetic aortitis. There was no apparent involvement, on gross or microscopic examination, of the aortic valve.*

In the second case, a man aged 43 years, the manifestation of superior vena caval obstruction was of recent and rapid, painless onset. He was seized with paroxysmal dyspnœa at 10.00 a.m., January 18, 1943, and exhibited the signs of partial superior vena caval obstruction afterwards. He had a loud systolic murmur and a blowing, high-pitched diastolic murmur — "almost continuous" wrote one experienced observer —heard best in the third left interspace. A tender, pulsatile liver was detected by several examiners. An autopsy seven days later revealed rupture of a syphilitic aneurysm of the ascending aorta into the auricle of the right atrium.

SUMMARY

A case of aneurysm of the aorta due to syphilis in a woman aged 44 years, with rupture into the superior vena cava, has been described. The clinical features of interest in arriving at the diagnosis were the explosive, painless onset of superior vena caval obstruction, the loud continuous murmur over the fistula, and the positive serological evidence of syphilis. An exact anatomical diagnosis was made by employing the technique of venous catheterization. This investigation revealed the interesting observation of mixing of the arterial and venous streams in both directions. Thirty-seven other cases of aneurysm of the aorta due to syphilis in the hospital records over a twelve-year period were reviewed. Two of these cases are briefly described: in one, a woman aged 51 years, the aneurysm had ruptured into the superior vena cava; in the other, a man aged 43 years, the aneurysm had ruptured into the auricle of the right atrium. In the second case, a pulsatile liver was present. A correct ante-mortem diagnosis of fistula had not been made in either case.

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* Information through the courtesy of Professor William Boyd, Department of Pathology, Banting Institute.

* Information obtained through the courtesy of Dr. W. G. S. Johnston, Toronto East General Hospital, where the patient died.

SPECIAL ARTICLE

THE REMOVAL OF SUPERFLUOUS HAIR BY X-RAYS

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Vancouver, B.C.

During the autumn and winter of 1930-31 advertisements appeared in the Vancouver daily papers, under the name of the Marton Laboratories, of which I had not heard before, offering removal of superfluous hair from the face. To any dermatologist or radiologist it was obvious from the wording that the method employed could only be x-rays. A booklet was obtained on application, which was published in the United States, in which it was stated that recent discoveries had made the use of x-rays for this purpose perfectly harmless, and the advertiser guaranteed perfect results which would be permanent. The renting of additional space in the building after a few months, and the fact that many prospective clients found that they had to wait several days for an appointment was evidence of a growing business.

I drew the attention of the Vancouver Health Department to the operations of this concern, and an investigation followed. It was learned that a regular business license had been issued by the license inspector, but the Health Department had not been consulted or informed of this. When the grave dangers to the public health were explained to him, as arising from this situation, the medical health officer became intensely interested. He infected the city license inspector with his zeal to eradicate this public nuisance and danger. Provided with a list of headings for an inquiry they made a descent upon the proprietor of the "Laboratories", in whose person was combined operator, technician and staff.

She produced no certificates or other qualifications testifying to her competence to operate x-ray apparatus. In lieu of this she stated that she had at one time been employed in a dentist's office in New Zealand, and in this office was an x-ray machine with which she admitted she had had nothing to do. A woman from the United States had for some months done similar work in a local apartment hotel, gave the present proprietor two or three months' instruction, sold (leased?) her the machine and departed whence she came.

Questioned about the machine and her technique, the proprietor had no idea what voltage she was using, said the amperage was "4", the distance between tube and skin "about 6 or 8 inches", the time of exposure "about 2 or 3 minutes", the size of each area treated was "about 4 by 4½ inches"; 10 to 20 treatments at intervals at first of 1 to 10 days and later 2 to 4 weeks constituted a "course". A filter was used, the thickness and composition of which was unknown. This was the secret of a "Doctor Marton of Paris". The name of the maker of the machine could not be learned, and there was no certificate of calibration or knowledge whether it had been calibrated.

The proprietor told the investigators that the Marton Laboratories had been operating in San Francisco and elsewhere in California and also in the State of Washington with full consent and approval of health authorities there. Statements were obtained by me from the Department of Public Health of the City and County of San Francisco, and the Board of Medical Examiners of the State of California to the effect that the Tricho System—which I then presumed to be the same concern under another name—had been "run out" of San Francisco; that the Health Department had never authorized it and would prevent its use if they knew of any place where it was being installed. The Board of Medical Examiners further submitted details of actions taken by them in the State of California in this connection, where the apparatus had been sublet to various beauty-parlours, etc., in charge of inexperienced operators. Further, law-suits had already been commenced in California against these operators for damage to elastic tissue, causing wrinkling and other disfigurements.

The above facts, together with an explanation of the dangers arising from the use of x-rays for removal of superfluous hair, especially in the hands of inexperienced operators, were submitted to the City Council of Vancouver. The medical health officer informed me that as a result of this steps would be taken at once to cancel the license of the Marton Laboratories. The proprietor of the concern began calling me frequently on the telephone asking for an interview, and finally one morning a woman appeared in my waiting-room announcing that she had come from Seattle for an interview in this connection, and that she would not leave until she got one. She sat it out until mid-afternoon and then disappeared.

At this point the city solicitor intervened with the announcement that there was no city statute under which the license of the Marton Laboratories could be revoked, cancelled or otherwise interfered with for such reasons as we presented. No laws being broken so far as the city was concerned the business might go on. It did.

About this time the Marton Laboratories was absorbed by a well-known beauty parlour which now announced its new name as the Arnold Dermic Laboratories. I took the matter up with the Provincial health officer and met with the same discouraging situation. So far as the Provincial laws were concerned

the business of destroying women's skins might continue. This officer was interested enough however to suggest that the Provincial Medical Act might be concerned. The legal advisers of the College of Physicians and Surgeons of British Columbia on consultation found that in treating by x-rays to remove a blemish, *i.e.*, superfluous hair, the Marton Laboratories or Arnold Dermic Laboratories was practising medicine within the meaning of the Act. It was only necessary to secure one or more witnesses or other tangible evidence to open a prosecution.

As the only acceptable witness would be a woman who had received treatment, or the only evidence which would be received would be an itemized and receipted bill, prosecution was of necessity delayed. Few women will go on the stand to admit that they have been dupes of a quack. Financial transactions were on a C.O.D. basis and no receipts were given.

For months past women had been encountered in private practice complaining of dryness of the skin and showing varying degrees of erythema, early atrophy and telangiectasia on cheeks, chin, nose and neck. Few had any suspicion of the cause of their trouble, but questioning invariably elicited the information that they had been treated at the Marton Laboratories from one to three years earlier. It was reported to me by one young woman that "over fifty" of her fellow-employees in a large department store had received treatment at the Marton or Arnold Laboratories, and had been given a special reduced rate of $75.00 for a course of treatment. Even allowing for exaggeration it is evident that these operations were being carried out on a wide scale.

It was considered wise to tell these patients to what they owed their trouble and to inform them that the changes in their skin were irreversible. Without dwelling upon the dangerous character of the possible sequelæ, each patient was urged to report at frequent intervals, and at once if warty growths appeared. With rare exceptions this advice has not been followed. It was apparent that many, especially those with incipient radiodermatitis, did not take the advice and comments seriously. Others, greatly upset, vigorously denounced not only the commercial firm concerned, but also the medical profession for permitting such a situation to exist. Others, "more than

Fig. 1.—Mrs. G.R.S., radiodermatitis of cheeks, nose and chin and squamous cell carcinoma of nose following treatment with x-rays for removal of superfluous hair.
Fig. 2.—Mrs. T.C.W., radiodermatitis of forearms and hands and squamous cell carcinoma right middle finger necessitating amputation following treatment with x-rays for removal of superfluous hair 17 years previously.

willing'' to help, stopped short at the suggestion that they could only do so by incurring some personal publicity. Nearly three years elapsed before a vigorously outspoken woman —a school teacher whose language was more forcible than ''parliamentary''—agreed readily to appear in court as witness for the prosecution.

The case came up in due time and with very little difficulty a verdict was obtained. A trivial fine was imposed, together, it is believed, with a ''cease and desist'' injunction from the Court. I anticipated that the fine would be recouped from the fee for the first case treated next morning, and that the business would go on, disregarding successive prosecutions, convictions, fines and injunctions until, if we were lucky, an impending jail sentence for contempt of court would wind the matter up. To my great surprise and pleasure however, the Arnold Dermic Laboratories closed out within a week, the beauty-parlour moved to another street and dropped the ''Dermic Laboratories'' and the x-ray machine.

A decreasing number of patients showing radiodermatitis from this source have continued to appear through the years since then. These have shown progressively later sequelæ: dryness of the buccal mucosa, dry lips, pronounced xerosis and scaling, indurated smooth white patches, and diffuse and local hyperkeratosis. The ultimate malignant sequelæ began to appear about 1945. Of these a few illustrative cases are cited.

CASE 1

Miss E.A., telephone operator, aged 36, seen in 1944, had been treated at the Arnold Dermic Laboratories in Vancouver in 1934. The skin of lips, chin and cheeks was finely wrinkled, with discrete, small atrophic patches, and numerous telangiectatic vessels. There was a circumscribed, indurated area in the left upper lip near the midline, with irregularity of contour of the vermilion border as if drawn up by contractile scarring or scleroderma. About the end of 1945 this area was resected for plastic repair, but not because of change in the epidermis suggesting cancer. Histologic study of the excised skin showed baso-squamous carcinoma.

CASE 2

Mrs. G.R.S., housewife, aged 43, seen in 1944, had had treatment at the Marton Laboratories ''several years'' previously. There was some shrivelling and telangiectasia at the tip of the nose and chin, and several minute keratoses on the chin. The skin of the cheeks, sides and back of the neck showed the same characteristics of radiodermatitis. There was a small shallow ulcer at the tip of the nose caused by accidental burn in an explosion 3 years before, which had not healed. With the use of a cod-liver oil ointment it healed in 5 months. In 7 months it reappeared with infiltrated margins and greater dimensions. An excision biopsy and plastic repair was done. Sections showed squamous-cell carcinoma.

CASE 3

Mrs. T.C.W., housewife, aged 59, seen in 1947, was treated in 1930 at the Marton Laboratories in Vancouver for superfluous hair of face, neck, extensor aspect of forearms and dorsum of hands. She presented typical radiodermatitis of the cheeks, sides and front of neck as far down as the angle of Ludwig, with pronounced atrophy of skin and soft tissue and numerous keratoses. The dorsal aspect of the middle phalanx of the right middle finger was occupied by a large ulcer with indurated, elevated and everted margins, the floor indurated, irregular and bathed in pus. This had gradually developed from a hyperkeratosis in the past two years. The finger was amputated through the distal extremity of the metacarpal. Regional lymph-nodes were not enlarged. Histological examination of the ulcer showed it to be a squamous-cell carcinoma.

CASE 4

Mrs. P.H.E., housewife, aged 43, seen in 1943, had been treated at the Marton Laboratories in Vancouver about 1928. She presented a chronic dermatitis of the arms, scalp, sternal and submammary regions and lower portions of the breasts. The same condition was present on the external aspect of the forearms and dorsum of the hands. She did not at this time reveal the fact that she had had x-ray treatment, and secondary infection was so severe and extensive that the true underlying situation was not at first apparent. It appeared to be a secondarily infected dermatitis supervening upon pruritus of undetermined origin which for years had been subjected by the patient to a numerous variety of home-remedies, proprietaries, etc. As this condition cleared under treatment it was found to be most persistent on forearms, hands and lower half of the breasts, where general atrophy, sclerosis with keratoses and telangiectases now became apparent. She then disclosed the history of treatment for superfluous hair on the forearms, in 1928, and of the appearance of the first symptoms of radiodermatitis on forearms and breasts in 1935. In treating the forearms the operator had crossed them over the body and in doing so the breasts had been pushed upward bringing their lower dependent parts much closer to the tube than the arms were. As soon as the acute infectious stage of the dermatitis was under control the patient disappeared. Three years later she was encountered again when she presented herself at my clinic at the British Columbia Cancer Institute on account of an ulcer which had first appeared on the lower part of the left breast, 2 years before, a year after she had left my care. She had treated it with the medicines which I had given her for the dermatitis, then resorted again to home and proprietary remedies and finally to a quack. She now had a huge and deep ulcer crater occupying practically the whole lower half of the breast and the chest wall immediately below it. Biopsy was hardly necessary to prove a squamous-cell carcinoma. Widespread metastases had already made their appearance and she died 4 months after she first came to the Cancer Clinic and 18 years after she had been treated by the Marton Laboratories.

The Bureau of Investigation of the American Medical Association which I approached recently for data in its possession referring to the ''Marton Method'' stated that very little had been heard recently of this concern. A reprint of an article in an old edition of its pamphlet dealing with cosmetics was very kindly supplied however. From this it appears that Jules Marcel Marton, ''not a graduate of any reputable medical school, or licensed to practise medicine anywhere in the United States'' had exploited this method of x-ray epilation under such names as ''Epilax-Ray,'' ''Dermic Institute,'' ''Hamomar Method'' and ''Hair-X Laboratories'', and doubtless many others. A pamphlet also put out by the Bureau of Investigation in 1930 listed a series of 56

cases reported by various dermatologists in eastern American cities, of radiodermatitis of varying degrees of severity following Tricho treatments some years before. None of these had as yet developed cancer. From the foregoing observations as well as those which follow it is not unreasonable to expect that "institutions" and "laboratories" of the same character will continue to make their appearance under new guises or disguises in other parts of the United States and Canada.

Recently Cipollaro and Einhorn[1] have published an article on the dangers inherent in the use of x-rays for treatment of hypertrichosis. This deals with the Tricho System chiefly, evidently a forerunner of the Marton Laboratories, describing in detail its origin and history, and recounting briefly the history of 66 patients observed by the authors and other dermatologists, who have suffered in degrees varying from chronic radiodermatitis to malignancy, many of which have probably since terminated fatally. The authors quote the statement which appears in the well-known textbook by MacKee and Cipollaro:[2] "it is now the consensus of dermatologists and roentgenologists that superfluous hair cannot be permanently removed with radiation of any kind, regardless of technique, without permanent injury to the skin". They also refer to the issue of a patent by the U.S. Patent Office to one Violet Arnold—*Nomen omen* (see above) —for an x-ray razor. This apparatus actually is a low-voltage, lightly filtered x-ray apparatus, for removing hair "without injury to the body tissues". It has already received considerable publicity in periodicals read by the lay public. That it cannot be efficient in any degree whatever and remain safe is evident from the statement by MacKee and Cipollaro quoted.

That many municipal and state public health authorities have remained unaware of the existence of the Tricho Institute, the Marton Laboratories and the x-ray razor and other such institutions and apparatus now being advertised, or due to appear, and the threat which this existence implies, is very apparent. The two concerns mentioned have been abolished, as far as I know, and for the time being, in California and British Columbia, but it is also a fact, as has been learned from conversations with patients, perusal of newspaper advertisements, etc., that they are still operating, in fact some have recently opened, in various cities in Canada and the United States. A dermatologist of eminence in a large eastern city in the United States told me a few months ago that the Tricho System which had been established in that city was, as far as he knew, still going, that he knew of one case of carcinoma of the jaw produced by such treatment, and had seen another case of extensive, diffuse radiodermatitis of both legs following such treatment recently by a Tricho system in a neighbouring city.

It would appear that dermatologists in the United States and Canada have been remiss in not bringing this matter to the attention of the public health authorities in their respective countries. There can be but few of them who have not encountered such cases as have been referred to, and who are not aware of the existence of commercial concerns of this sort who are thus operating to the public danger. No group of physicians is better informed of the dangers of indiscriminate exposure of human skin to x-rays. It is the duty of this group to inform the public health authorities fully of the facts, and when necessary to bring pressure to bear in assisting them to see to it that legislation is enacted and enforced which will make it impossible for these dangers to continue.

SUMMARY

Observations of cases of radiodermatitis on various parts of the face, limbs and body, resulting from x-ray treatment of hypertrichosis by a commercial institution, made over a period of 17 years, and an account of successful legal action taken to end such operation in British Columbia, are related.

Four illustrative cases from a series of malignant sequelæ observed, one necessitating amputation, and one terminating fatally, are described.

The view that permanent removal of superfluous hair by radiation therapy cannot be accomplished without permanent skin injury is strongly supported.

Removal of superfluous hair by roentgen therapy is still being widely practised by commercial concerns and constitutes a menace to which public health authorities are not giving adequate attention.

A preliminary report on the activities of the Marton Laboratories in Vancouver was published by the present author in the *Canadian Medical Association Journal* (**24**: 847, 1931) and I am indebted to the Editor for permission to republish portions of that article in the present paper.

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Make it an invariable and obligatory law to yourself never to mention your own mental diseases. When you talk of them, it is plain that you want either praise or pity; for praise there is no room, and pity will do you no good.—Johnson.

CLINICAL and LABORATORY NOTES

REPLACEMENT TRANSFUSION IN ERYTHROBLASTOSIS FETALIS

(Report of 24 Cases)

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During the past decade erythroblastosis fetalis, with its clinical manifestations of progressive anæmia, jaundice, enlarged liver and spleen in the newborn has been established as due to the sensitization of maternal Rh negative blood by either a transfusion of Rh positive blood or by an Rh positive fetus.[1] Recognition of the pathogenesis of the disease has led to its treatment by transfusion with Rh negative blood. It is unlikely that Rh positive blood survives long enough to combat the anoxia which is present. Removal of the child's blood and replacement with Rh negative blood would appear to be ideal inasmuch as one might remove deleterious isoagglutinins and at the same time replace oxygen-poor erythroblasts with mature red cells. Replacement should be done soon after birth in order to lessen irreversible changes in tissue such as brain, kidney, and liver.

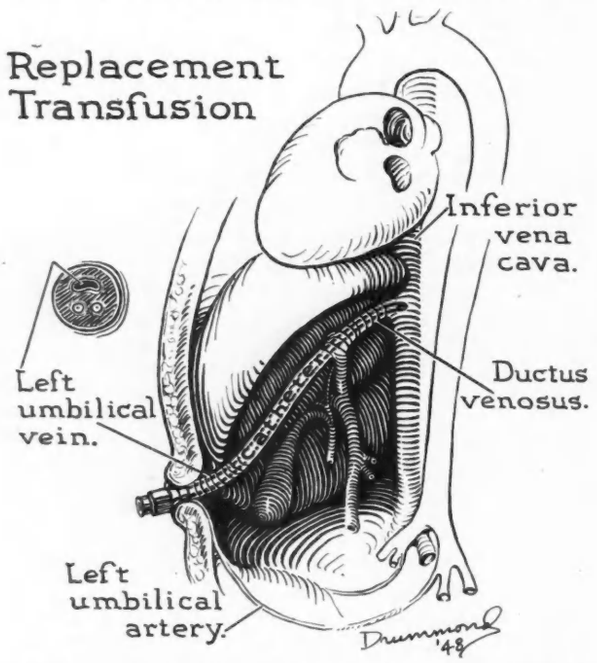

Fig. 1 illustrates the catheter in place in the umbilical vein.

In the past year we have treated 24 cases by replacement transfusion, rather than repeated transfusions, in an effort to evaluate this form of therapy. Exsanguination, or replacement transfusion is not a new procedure. It was first reported from this hospital in 1921 by Robertson, Brown and Simpson,[2] and again in 1925 when Hart,[3] suggested its use in erythroblastosis fetalis. We have followed the lead of Diamond,[4] and have used the umbilical vein, which remains patent several days post-partum. The procedure is simple but must be carried out with extreme care. Rh negative stored blood (as fresh as possible) in amount equal to 100 c.c. per pound of body weight should be available. The infant must be kept warm and oxygen administered almost continually. The operator should wear a stethoscope which is strapped over the infant's præcordium. The umbilical vein is catheterized with a woven venous cannula, (size 6, C. R. Bard, Inc.); 20 c.c. of blood are withdrawn from the vena cava and immediately replaced with 20 c.c. of the stored blood. This is repeated slowly allowing 5 minutes for removal and insertion of the blood until 100 c.c. per pound is replaced, leaving a positive balance of 15 c.c. per pound

TABLE I.
REPLACEMENT TRANSFUSIONS
24 CASES

Age on admission	1 hr. to 4 days
Average birth weight	6 lb. 3 oz.
Jaundice present	22 cases
Enlarged liver	21 "
Enlarged spleen	21 "
Hæmoglobin on admission	4.2 to 16.2 gm.
Average	9.8 gm.
Red blood count on admission	1.1 to 5.3 million
Average	2.9 million
Patients' blood	
Rh positive	24 cases
Group O	11 "
Group A	8 "
Group B	3 "
Group AB	2 "
Erythroblasts in smears 13-85 per 100 w.b.c	17 "
Antibodies present	20 "
Mothers' blood	
Rh negative	24 "
Antibodies	24 "
Fathers' blood	
Rh positive	24 "
History of miscarriage or stillbirth	14 "
Previous transfusions	3 "
Blood used for replacement	
Rh negative	24 "
Group O neutralized	3 "
Amount removed	130 to 720 c.c.
Average	440 c.c.
Amount replaced	180 to 750 c.c.
Average	480 c.c.
Percentage replacement	55 to 95%
Complications—Cardiac embarrassment requiring supportive treatment	5 cases with 1 death
Hæmoglobin on third day	9.5 to 18.1 gm.
Average	12.8 gm.
Supportive transfusions 50 to 150 c.c.	18 cases
Days in hospital	6 to 35
Average	12
Deaths due to transfusions	
Cardiac failure	1
Deaths due to erythroblastosis	1
Deaths due to intestinal intoxication 20 days postoperative	1

of body weight. To prevent clotting, ½ c.c. of heparin solution (1 c.c. to 100 c.c. of saline), is used in each 20 c.c. of blood and frequent samples are taken to estimate the clotting time and percentage replacement. Great care must be exercised in both removal and replacement of blood in an infant whose total circulating blood volume is not more than 300 to 400 c.c. The appearance of a murmur, undue slowing or acceleration of the heartbeat are indications to proceed more slowly. Haste may result in cardiac embarrassment and the procedure should take from one to two hours.

In our series of 24 cases we have had 3 deaths, one from cardiac embarrassment, one from intestinal intoxication at 20 days and one from erythroblastosis fetalis of the kernicterus type. The last patient was moribund on admission and at post mortem showed brain damage associated with kernicterus.

In conclusion we suggest that replacement transfusion in the treatment of erythroblastosis fetalis has given the best results to date.

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ON THE DETECTION, DIFFERENTIATION AND TITRATION OF ANTI-RH ANTIBODIES*

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Anti-Rh antibodies occur in at least two states or forms. For the detection of the presence of one of these in a serum, red blood cells suspended in saline are ordinarily used; for the other, plasma, serum, albumen or other colloid must be added. The former are variously termed agglutinins or saline, early, immature, bivalent or thermolabile antibodies; the latter glutinins or partial, incomplete, coating, blocking, inhibiting, hyperimmune, late, mature, monovalent or thermostabile antibodies. We do not known for certain how the one differs from the other, why one rather than the other develops in a given patient at a given time, or that the one kind rather than the other is more likely to produce disease.† One thing we do know about them is this difference in the mode of demonstrating them. We have, therefore, in our laboratory come to use these qualities to designate them, naming them respectively "saline" and "albumen" since an albumen solution is the colloid we use.

Some investigators, notably Hill and Haberman,[1] have brought forward evidence for the existence of several other forms of Rh antibodies. While this may prove to be true, it has not yet been verified nor its clinical importance established, so that, for the purpose of this paper but two forms will be considered.

DETECTION OF ANTIBODIES

We have now examined the sera of nearly 4,000 Rh-negative women and, having tried various methods for the detection of antibodies, have concluded that for first demonstration the simplest is the albumen suspension method, which is included in Diamond and Denton's[2] paper on the use of albumen in agglutination. This will detect the presence of Rh antibodies whether saline or albumen, but will not differentiate them. In our hands it has proved as sensitive as, less time-consuming and less open to error than, that of Wiener, whether in the original form[3] or its variants up to and including the current one.[4] We have entirely given up Diamond and Abelson's[5] slide method. While Dr. Diamond has many times demonstrated this test to one or other of us in his laboratory and there it has been clear-cut, in our own this has not always been so; on several occasions the method has failed to pick up antibodies demonstrable by other methods. We have not had an extended experience with Berlin's[6] adaptation of our capillary method. Our present impression is that it is open to major errors, though further experience may prove us wrong. The method of Coombs, Mourant and Race[7] we use practically only for evidence of sensitization of the cells of the newborn.

Albumen suspension method:

Materials:

1. Test tubes approximately 7.5 cm., long; inside diameter 0.8 cm.; smooth, round bottom.
2. One c.c. serological pipettes; a separate pipette for each serum.
3. Thirty per cent bovine albumen.*
4. Normal saline.
5. Red blood cells. Rh-positive and Rh-negative cells are required. They should preferably be group O, but if a single serum is being tested they may be of the same group as the serum. The cells must be fresh—not over two days old—and must be kept in a refrigerator. Our own practice is to use for our Rh-positive

* From the Children's Hospital and Department of Pædiatrics, University of Manitoba Medical School, Winnipeg. Assisted by a grant from the Associate Medical Committee, National Research Council, Ottawa.

† We make these statements categorically, knowing that some investigators believe they do know these things. This paper is, however, not written as an argument but as a simple statement of practical laboratory methods found useful in a field in which there is much confusion.

* Supplied by Armour Laboratories, Armour & Co., Union Stock Yards, Chicago, Ill. We have recently made use of 25% human albumen kindly supplied by Dr. W. P. Boger of Sharpe and Dohme. This has given identical results with the bovine albumen.

cells, cells of phenotype R_1R_2,* from one donor whose blood has been found to have excellent agglutinability. The agglutinability of cells varies greatly, and can be determined by setting up parallel titrations with cells from different donors. It will be found that some give a more clear-cut and easily read reaction than others. The Rh-negative cells must be of genotype rr. It is wise to use cells from two Rh-positive and two Rh-negative donors. Just before use enough cells for one day's tests are washed three times in normal saline, and, after pouring off the last saline, enough of the washed cells added to the 30% albumen to make a 2% suspension—usually about 2 drops of the washed cells to 1 c.c. of the 30% albumen. The Rh-positive and the Rh-negative suspensions are made up separately.

6. The serum or other fluid to be tested. This must be sterile. Contaminated specimens sometimes give false positive and sometimes false negative results. The sera should also be fresh. Some sera stored in the refrigerator at approximately 4° C. retain their original titre for many months: others deteriorate in a few weeks or months. The latter is particularly true of saline antibodies. Demonstration and titration are, therefore, a record of antibody only for the day the examination was carried out, and do not necessarily represent the state of affairs at the time the serum was drawn. Every serum is examined whole and also diluted 1 in 16 with saline. The need for examining sera in dilution as well as whole, when seeking for Rh antibodies, was first pointed out by Taylor *et al.*[8] This is true of the present albumen suspension method as it is of other methods.

Method.—For every serum to be tested number six of the small test tubes, place them in a rack and load as follows:

Tube 1. 2 drops whole serum under test.
2 drops albumen-suspended red blood cells from Rh-positive donor No. 1.

Tube 2. 2 drops whole serum under test.
2 drops albumen-suspended red blood cells from Rh-positive donor No. 2.

Tube 3. 2 drops serum under test diluted 1 in 16; red blood cells as in tube 1.

Tube 4. 2 drops serum under test diluted 1 in 16; red blood cells as in tube 2.

Tube 5. 2 drops whole serum under test.
2 drops albumen-suspended red blood cells from Rh-negative donor No. 1.

Tube 6. 2 drops whole serum under test.
2 drops albumen-suspended red blood cells from Rh-negative donor No. 2.

Shake the tubes vigorously; let them stand at room temperature for ten minutes, then centrifuge. The time and speed of centrifuging must be learnt by experience, —a sharp-edged button of cells in the tube, not unduly packed, is required. Using a fully loaded No. 1 S.B. International Centrifuge with rheostat at 15, two minutes from "Turn on" to "Turn off" gives good results.

To read macroscopically the tube is agitated a little and tilted almost horizontally several times.

* We will be glad to determine the phenotype of cells for other laboratories not in a position to do this themselves. For this purpose 1.0 or more c.c. of sterile, clotted blood is required.

We record the degrees of agglutination as follows:

S	Solid agglutination, a button of cells floating in clear fluid.
++++	Large, firm clumps of cells suspended in clear fluid.
+++	Moderately large clumps of cells with slightly cloudy fluid due to a small number of unagglutinated cells.
++	Small but distinct clumps with cloudy fluid. When viewed under the low power of the microscope there are several moderate-sized tight clumps of cells to each field.
+	Merest suggestion of finest dust macroscopically. Under low power there are quite a few tight clumps of two to five cells in every field.
±	Doubtful. Under low power there are groups of two, three or four cells sticking together, but no definite agglutinates.
–	No agglutination seen microscopically.

Occasionally, readings of +++±, ++±, etc., are used for intermediate reactions. Tubes read as solid, ++++, +++ or ++ are not checked microscopically but all others are. A reading of ± is not considered positive. The titre of the serum is taken as the highest dilution in which it gives at least a + agglutination. For microscopic reading some of the mixture is taken on the end of a glass rod, mixed with a drop of saline on a slide, allowed to stand one to two minutes and then examined. As already stated, this method detects the presence of Rh antibodies, whether they be of the saline or of the albumen type, but does not differentiate between them. If all one wants to know is whether or not an antibody is present one need go no further. Usually one will want to know at least the strength of the antibody. Titration is carried out by diluting the test serum serially with normal saline, and proceeding as above with each dilution.

Detection and titration of saline antibody.—We have found, contrary to our earlier statement,[9] that the capillary method can be used for the detection as well as for the titration of saline antibody. The reaction is precise and much easier to read than the test tube methods. The key to its successful use in detection is the thorough washing of the red cells; to give titration levels essentially identical with those obtained in the test tube, final readings are made at 60 to 75 minutes, although first agglutination may take place in 10 minutes or less in the higher strengths.

The same sort of Rh-positive cells are used as for the albumen suspension method. They are washed three times in at least twenty times their volume of normal saline, and are finally suspended in saline to give an approximately 20% suspension. (Some of the suspension is spun down in an hæmatocrit tube to check its

strength). The test is carried out as described for Rh typing.[9] Titration is done by serially diluting the serum with saline, setting up each dilution with the test cells, and reading the whole at 60 to 75 minutes. The agglutinates in the endpoint capillary will be very fine.

Interpretation as to type and titre of antibody.—If one gets agglutination by the albumen suspension method it is certain that there is an Rh antibody in the serum. If one gets agglutination in the capillary under the above conditions, it is certain that there is a saline antibody present. If one gets agglutination by the albumen method and not by the capillary, there is an albumen antibody present. If one gets agglutination in both, then the type or types of antibody present can only be decided by titration. To put this briefly:

Agglutination by albumen method—Rh antibody present.

Agglutination in capillary—Rh saline antibody present.

Agglutination in albumen only—Rh albumen antibody present.

Agglutination in both—decide by titration, thus:

1. Titre in capillary and in albumen suspension identical or only one dilution apart: interpret as pure saline antibody.

2. Titre in capillary several dilutions less than in albumen suspension: interpret as two antibodies present, saline antibody of the capillary titration titre, albumen antibody of the albumen suspension titration titre. If the albumen titre is very strong, say 1:512, and the saline only demonstrable with undiluted serum, the latter may be due to a separate antibody, or possibly to an effect of the strong albumen antibody.

3. Titre in capillary more than one dilution stronger than in the albumen suspension. This occurs rarely. Probably best interpreted as pure saline antibody. These are best illustrated by actual examples:

	Dilution									
Method.....	1	2	4	8	16	32	64	128	256	512
Capillary.....	+	+	+	+	+	+	+	+	+	
Albumen.....	S	S	S	++++	++++	+++	+++	++	+	

Interpretation:—Saline antibody only.

	Dilution									
Method.....	1	2	4	8	16	32	64	128	256	512
Capillary.....	+	+	+	+	+					
Albumen.....	S	S	S	S	S	++++	+++	++	++	

Interpretation:—Saline antibody, titre 1:16
Albumen antibody, titre 1:256

	Dilution									
Method.....	1	2	4	8	16	32	64	128	256	512
Capillary.....	+	+								
Albumen.....	S	S	S	++++	++++	+++	++	+		

Interpretation:—Albumen antibody, titre 1:128
Probable saline antibody, titre 1:2.
Capillary reaction may possibly be due to the albumen antibody.

	Dilution									
Method.....	1	2	4	8	16	32	64	128	256	512
Capillary.....	+									
Albumen.....	S	S	S	S	++++	++++	++++	+++	+++	++

Interpretation:—Albumen antibody, titre 1:512
Saline antibody, doubtful. Reaction in capillary probably due to the albumen antibody.

Specificity of Rh antibodies.—There are, as is well known, several Rh antibodies of different specificity.[10] To determine the specificity of any serum one must have available blood cells of several Rh phenotypes. Since these are available in only a few laboratories, of which ours is one, we will be glad to determine the specificity of sera for any other Canadian or American laboratory. It does not seem worth while to set out the method here.

Summary

For the detection, differentiation and titration of Rh antibodies two fairly simple methods are recommended. For first detection the sera being investigated are added both whole and in a 1 in 16 dilution to suspensions of Rh-positive and whole to suspensions of Rh-negative cells in bovine albumen and, after every brief mixing and centrifuging, the sediment examined for agglutinates. For differentiation, titration is done by this method and also by using saline-suspended cells mixed with the sera in capillary tubes. The methods and interpretations are given in detail.

We gratefully acknowledge our debt to the Associate Medical Committee, National Research Council, Ottawa, for their support of the investigation upon which this report is based, and for their permission to publish this paper, and to Drs. L. K. Diamond, J. Hill and S. Haberman for supplying us with some of the rarer anti-Rh sera.

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EDITORIAL

SALT AND WATER DEPLETION

THE Croonian Lectures delivered in London in December, 1946, by Dr. H. L. Marriott* deal exhaustively with the physiology, clinical manifestations and treatment of water and salt depletion in the human subject. These articles will be worthy of close reading but for the moment we will refer only to the sections of more direct clinical application.

The important distinction is made between dehydration, the result of water depletion, and dehydration, the result of salt depletion. The two conditions, although often associated, differ from each other in mechanism of production, symptomatology and treatment indicated. When dehydration is recognized, every effort should be made to ascertain whether it is due to salt depletion, water depletion, or a combination of both. The administration of saline solution to patients suffering from "pure" water depletion makes them worse and may be fatal. On the other hand, an essential condition in the causation and aggravation of "pure" salt depletion is a liberal intake of unsalted water. If salt depletion goes unrecognized and terminates fatally, the death may be ascribed to "toxæmia", "uræmia" or "circulatory failure".

Salt depletion almost always arises from excessive losses of sodium chloride in sweat, in intestinal secretions (vomiting; diarrhœa; prolonged gastric or intestinal suction; biliary, intestinal, or pancreatic fistulæ), or, in Addison's disease, in the urine. Rarely in temperate climates is the salt intake so low that the body's power of salt conservation is unable to maintain the salt balance of a healthy person. Symptoms of pure salt depletion include lassitude, muscular weakness, headache, anorexia, orthostatic giddiness and fainting, muscular cramps, nausea, vomiting, apathy, and mental confusion and delusions. Weight is lost and the patient appears dehydrated. The plasma volume drops and hæmoconcentration occurs. The blood viscosity is increased. The plasma sodium and chloride fall and the urea rises. When there is disproportionate loss of sodium chloride ions—such as the predominant loss of sodium in diarrhœa and of chlorine in vomiting—the acid-base equilibrium may be shifted into either an acidosis or an alkalosis. Terminally, the blood pressure drops and the clinical picture is one of peripheral circulatory failure (oligæmic vasoconstrictive shock). In the last stages of depletion, a 70 kg. man may have a deficit of 6 to 10 litres of isotonic saline. Of special importance from the diagnostic standpoint are the following observations: (1) pure salt depletion, in distinction to pure water depletion, is *not* accompanied by thirst; (2) in patients with pure salt depletion the urine volume remains normal till late; (3) except in patients with Addison's disease, chlorides disappear from the urine early in the course of salt depletion.

Dr. Marriott emphasizes that for the successful treatment of salt depletion, not only must its presence be recognized, but an estimate must be made of its degree at the outset of treatment, and its progress during the course of treatment. As a general rule the severity of the symptoms and the presence or absence of chlorides in the urine (except in patients with Addison's disease) form a better guide to treatment than determinations of blood constituents. Isotonic saline solution (0.85%) should be given until symptoms are relieved and chlorides are consistently present in the urine. Thereafter hypotonic saline (isotonic saline plus 5% glucose solution) should be given. Hypotonic saline is recommended for use throughout the treatment of mixed salt and water depletion and for the treatment of infants. Initially, if symptoms are severe, the intravenous route should be employed. Apparently fluids are retained in the stomach for a long time in severe salt depletion and, if vomiting occurs, aspiration of the vomitus may lead to pulmonary complications. The amount of fluid given should be sufficient to restore the patient to a normal state of balance. The equivalent of 10 litres of saline or more may be required for an adult severely depleted. A good objective to work for in the treatment of an adult is a urine volume of not less than a pint (570 ml.) with a salt content of 3 to 5 g. per litre per 8 hour period. It is pointed out that during the intravenous administration of saline some sodium chloride may

* *Brit. M. J.*, 4493 (Feb. 15), 245; 4496 (March 8), 285; 4497 (March 15), 325, 1947.

spill into the urine even though the patient is still grossly salt deficient. In emergencies, intravenous injections of saline should be given rapidly: the first pint in 10 minutes; the second in 15 minutes; the third in 20 minutes; the fourth in 30 minutes; then a pint every 2 hours. Watch must be kept for signs of œdema, especially pulmonary œdema. Serious consequences may result from the administration of potassium salts or of copious amounts of unsalted water to patients depleted of sodium chloride.

The commonest clinical causes of pure water depletion are great weakness with disinclination to make the effort required for drinking, coma, and dysphagia due to œsophageal disease. Thirst and dry mouth are early and progressive symptoms. Later appear oliguria, weakness, an ill dehydrated appearance, slight personality changes, and, finally, marked impairment of mental and physical capacity; 15% of the body weight may be lost before death. The biochemical and circulatory changes do not appear so early nor do they tend to be as profound in water depletion as in salt depletion. The biochemical changes also differ qualitatively. The plasma and chloride may be elevated and, sodium and chloride continue to be excreted in the urine. In treatment, water can usually be administered by mouth or rectum. It is rapidly absorbed from the gastro-intestinal tract. If a parenteral route is necessary 5% glucose solution should be employed. The urine volume is the best indication of the amount of water required. The same objective should be sought as in cases of salt or mixed salt and water depletion, *viz.*, a pint of urine containing 0.3 to 0.5% sodium chloride per 8 hour period. The transnasal intragastric drip method of administering fluid is recommended for use in suitable circumstances.

Special care must be taken of patients who have water or salt depletion or both and are suffering from the effects of acute hæmorrhage, anæmia, hypoproteinæmia, capillary damage from anoxia or toxæmia, or damaged kidneys unable to secrete salt normally. Preliminary treatment with transfusions of blood or plasma, or concomitant treatment with oxygen or both may be required. Watch should be maintained most intently for the development of pulmonary œdema in patients of this kind. The author points out some of the peculiarities of renal function in infants and emphasizes that infants are particularly susceptible to dehydration and also, to superhydration due to too vigorous fluid therapy. Children are therefore, another group of patients deserving of most careful investigation and thought before and during fluid therapy.

EDITORIAL COMMENT

Snoring

There is almost no serious literature on snoring and yet few of the minor afflictions of mankind can be so hopelessly infuriating, even to the point of tragedy. Marriages may be broken up, and camping parties may be ruined by snoring. Can tragedy go deeper!

A recent study by Ian G. Robin (*Proc. of Roy. Soc. of Med.*, **41**: 151, 1948) shows that snoring may involve various structures in the respiratory tract, but these do not all necessarily take part in the noise. It is only in comparatively few people that to the ordinary rumble there is added the weird accompaniment of wheezing and whistling which presents such possibilities in humorous burlesque. These extra sounds are produced by the epiglottis, the tongue, cheeks, lips or nostrils. The present study however is concerned only with the sounds produced by vibrations in the soft palate and posterior faucial pillars. It has been proved by means of a pharyngoscope that the snore is produced by vibration of the thin edge or velum of the posterior faucial pillar. A reservoir of air in the nasopharynx is necessary. When a "critical point" or position is reached in the relative position of the tongue and soft palate, the velum vibrates. But in addition to the position, the production of the snore depends on the texture of the velum, which again is affected by the muscle tone of the glossopharyngeal arch and the thickness of the tissues, especially the mucosa. The position of the tongue has a part in the "critical moment". Sleeping on the back tends to produce snoring because the tongue falls back more readily then. Dentures seem to play very little part either way.

It is the tone of the musculature which appears to be the most important single factor. But just what produces alterations in this tone during sleep is still open to question. One theory is that there is a fall in adrenal secretion during sleep leading to a lowered tone of the palatal structures. Some lay stress on the psychological aspect, even to the point of suggesting that snoring is aggravated by worry. But at least some snorers are equally bad at all times. Snoring occurs mainly in mouth breathing, so that any nasal obstruction should tend to cause it, but all mouth breathers do not snore. Still, removal of nasal obstruction is helpful, as is also change of position. For those who will tolerate it a cotton reel sewn into the back of

the pyjamas is efficacious. Amputation of the uvula very seldom gives any relief, though it may change the pitch of the snore. Keeping the mouth closed by a special splint (the Andresen) or even by adhesive, is very helpful. Altering the texture of the pillars by sclerosing injections has been tried, but with only equivocal results. Possibly tonsillectomy helps by leaving a fixed fibrous band instead of soft mobile tissues.

When all is said, snoring is a symptom of unbalanced breathing which may be accompanied by either pathological or physiological conditions. The remedy must therefore fit the causes. Isolation may be the only effective measure.

MEN and BOOKS

ABRAHAM GESNER, M.D., SURGEON GEOLOGIST, 1797–1864*

Kenneth A. MacKenzie, M.D.

Halifax, N.S.

This is the story of a country doctor who practised in Nova Scotia over a century ago. His name has now a permanent place in the archives of the Maritime Provinces due to his work in geology which led him to invent a method of distilling an illuminating oil from coal. He practised medicine for twelve years while pursuing his hobby and it is fitting that we should place in our archives some record of his career and achievements.

Abraham Gesner was born at Cornwallis, Kings Co., Nova Scotia, in 1797. His father, Colonel Henry Gesner, a United Empire Loyalist, fought in the Revolutionary War; lost all of his worldly possessions; then moved to Canada where he was given a grant of four hundred acres of land in the Annapolis Valley. He married a Miss Pineo, and Abraham was their sixth child of a family of twelve. Abraham married Henrietta, daughter of Dr. Isaac Webster, of Kentville, and had eleven children, some of whom attained eminence in the learned professions—medicine, geology and theology. As a young man Gesner shared in a venture to sell horses in the West Indies. Two of his ships were wrecked, one at Brier Island where he almost lost his life by drowning. Later, he proceeded to London where he studied medicine and surgery, at St. Bartholomew's and Guy's Hospitals, under distinguished teachers including Sir Astley Cooper and John Abernethy.

PARRSBOROUGH PERIOD, 1824–1837

In 1824, Dr. Gesner returned to Nova Scotia and settled in Parrsborough, one of the most picturesque and interesting localities in the Maritimes. He gives one reason for his choice of location, "that there was an abundance of rocks and minerals to be studied during his leisure hours".

Gesner's medical work is completely overshadowed by his work on natural history, especially geology. Unfortunately he has left no record of his medical activities. A brief reference may be made here to the conditions of medical practice of his time and to the physiography of the district in which he did his pioneer work. It was before the days of anæsthesia and antisepsis, and there was no clinical thermometer. Medical practice consisted of simple diagnoses, empirical remedies, minor surgery, including fractures, extraction of teeth, and the management of difficult labour, when the midwife failed. The physician had ample time for indulging in "hobbies", if so minded, and golf had not appeared to divert the busy doctor from more useful forms of recreation. To visit his patients he either walked, or rode on horseback. Carriages were not known in his district. Railroads had not yet been built; country roads were little more than footpaths. One can picture Dr. Gesner visiting his patients on horseback with his saddlebag full of empirical remedies, probably home-made, and returning home with a saddlebag full of rocks and fossils.

The common illuminant for the home was the tallow candle, home-made, and "dips" made from seal oil, whale oil or vegetable oils. Dr. Gesner burnt the "tallow candle" instead of the "midnight oil".

The physiography of this district is one of the most interesting in the world. Parrsborough, then only a village, situated at the mouth of a river flowing into the Bay of Fundy, was flanked by beautifully wooded hills and magnificent cliffs. Opposite the village, across Minus channel nine miles away, arose the majestic headland of Blomidon. The Bay of Fundy is noted for its remarkable tides which rise and fall forty to fifty feet twice daily. The movement of huge bodies of water produce treacherous currents and constant erosions make fresh exposures of rock for hundreds of miles. This magnificent panorama of cliffs has been a source of pleasure to travellers and lovers of nature, and a nightmare to mariners, many of whom have been ship-wrecked on the treacherous coast line. Even the Indians were intrigued by the grandeur of the district and built up legends about the abode and activities of their gods. The legend of Glooscap is a familiar feature of Indian lore. To Gesner, however, these cliffs made a different appeal. In few places in the world have the pages of geological history been so beautifully and wonderfully exposed, and Gesner, the lover of nature, made the most of his opportunities. On the shore of his village he could pick up

* Read at the Seventy-ninth Annual Meeting of the Canadian Medical Association, Section of Historical Medicine, Toronto, June 25, 1948.

amethysts, opals, carnelian, agates, jasper, and other minerals from the trap rock—volcanic in origin—which had at some time pushed through the triassic sandstone. Every headland, gully and river bed for miles around was a fruitful field for the amateur geologist. Some miles away he studied the strata of the Carboniferous Period along the famous Joggins shore. For thirty miles along this shore may be seen seventy seams of coal separated by beds of sandstone, shale and slate, representing a thickness of ten miles. On the shore one finds magnificent fossils, giant trees, lepidodendra, cyads, ferns, calamites and sigilaria, many of which have found their way to, and may now be seen in many museums of North America and Europe.

Dr. Gesner practised for twelve years in this district and, as far as we know, his practice was his only source of income. During his leisure hours he explored the whole district, at first near his home, later in more remote parts of the country. He kept careful notes of his findings, and in 1836 he was able to complete a book which was published in Halifax by Gossip and Coade. It was entitled "Remarks on the Geology and Mineralogy of Nova Scotia", by Abraham Gesner, Surgeon. In his preface he apologizes for the imperfections in his work by stating, "that it has not been prepared with leisure and retirement. On the contrary, amidst the arduous duties of a laborious profession and under the annoyance of perpetual interruption, most of the pages have been written; or during the silent hour of midnight when the labour but not the fatigue of the day had departed". This statement indicated that he was still a busy practitioner. At this point, it should be noted that Dr. Gesner did this work alone. Geology was in its infancy. Darwin's work did not appear until thirty years later. The hypothesis of the Ice Age had not yet been advanced. Gesner, reputed to be a religious man, must have had many struggles to reconcile the evidence of his eyes with the biblical accounts of creation and the flood. One may surmise that he had many arguments with the theologians of his day.

SAINT JOHN PERIOD, 1838–1843

Gesner's book attracted immediate attention and brought him before the public as a geologist. In 1837, he explored certain regions in New Brunswick—Grand Lake, Salmon River and Richibucto. The following year he was appointed Provincial Geologist for New Brunswick, a position which he held for three years. He made three annual reports which are preserved. At the end of this period he wished to continue his explorations but the Legislature did not see their way clear to continue his services and he was stranded in Saint John without funds. At this time he was the possessor of a large natural history collection, said to number 4,000 specimens. Most of these were gathered in Nova Scotia while he was still in medical practice. They included animals, birds, reptiles, fish, Indian relics, as well as minerals, rocks, and fossils. He attempted to interest the Government in the formation of a museum but met with no success. He had an exhibition of his collection in Halifax, in 1841, and in Saint John, in 1842. His next move was to open a private museum in Saint John hoping that the admission fees would cover the expenses. He obtained a suitable room in the building of the Mechanic's Institute, issued a prospectus and prepared a catalogue. He was, by this time, hopelessly in debt, having borrowed money from prominent citizens of Saint John. He tried to sell his collection but found no buyers. Finally, his creditors agreed to accept his collection in payment. The Gesner museum was the first Natural History Museum in Canada, 1842, and was the nucleus of the present New Brunswick Museum which contains many of Dr. Gesner's original specimens.

In 1843, he returned to Nova Scotia and resumed medical practice at his old home, Cornwallis. He continued to write extensively on the natural resources of the Maritime Provinces, and carried out experiments on coal gas and coal oil which led him to fame.

KEROSENE PERIOD, 1846–1863

Gesner's important invention was a retort which enabled him to distil an oil from coal. The probable date of his earliest experiments is 1846 and the special type of coal used was Albertite found in New Brunswick; the patents were taken out some years later. In 1850, Gesner met at Halifax the Commander-in-chief of the British North American Station, Lord Dundonald, who is said to be the original discoverer of illuminating gas. He had a commission from the British Government to inquire into the possibilities of producing fertilizer, from the asphalt deposits in Trinidad, for the coffee and sugar plantations of the West Indies and Gesner was employed to assist him. This gave Gesner an opportunity to continue his oil researches with asphalt. The oil which he was able to produce from asphalt, and various types of coal, was a clear oily liquid which proved to have better illuminating properties than the gases already in use. Gesner coined the word "kerosene". At first he called it "Keroselain", from two Greek words meaning "wax" and "oil" and later contracted it to kerosene. He formed a company in Halifax which did not prove to be a success. In 1853, he went to New York, took out patent rights and sold them to the New York Kerosene Company. He remained there for ten years taking an active part in the construction of an oil plant at Hunter's Point, N.Y. The old Gesner refinery is still in existence, the

property of the Standard Oil Company. Discovery of natural oil wells in Pennsylvania, in 1859, produced cheaper oil and interfered seriously with the New York industry. In 1863, Gesner sold his interests and returned to Halifax to take the chair of Natural History at Dalhousie University. He died the following year, 1864.

This remarkable man's activities covered a wide field. He was in turn a farmer, trader, surgeon, geologist, author, inventor, manufacturer and lecturer. His writings covered a wider field than geology, he wrote on the fauna and flora of the country and was especially interested in the commercial possibilities of the natural resources. While in his earlier years he toiled alone, in later life he made contacts with distinguished men. In 1842, he conducted Sir Charles Lyell and J. W. Dawson along the Joggins shore. In 1840, he was made a Fellow of the Royal Geological Society of London. Later he was made a corresponding member of the Geological Society of Canada, Academy of Natural Sciences of Philadelphia, and the Geographic Society of New York.

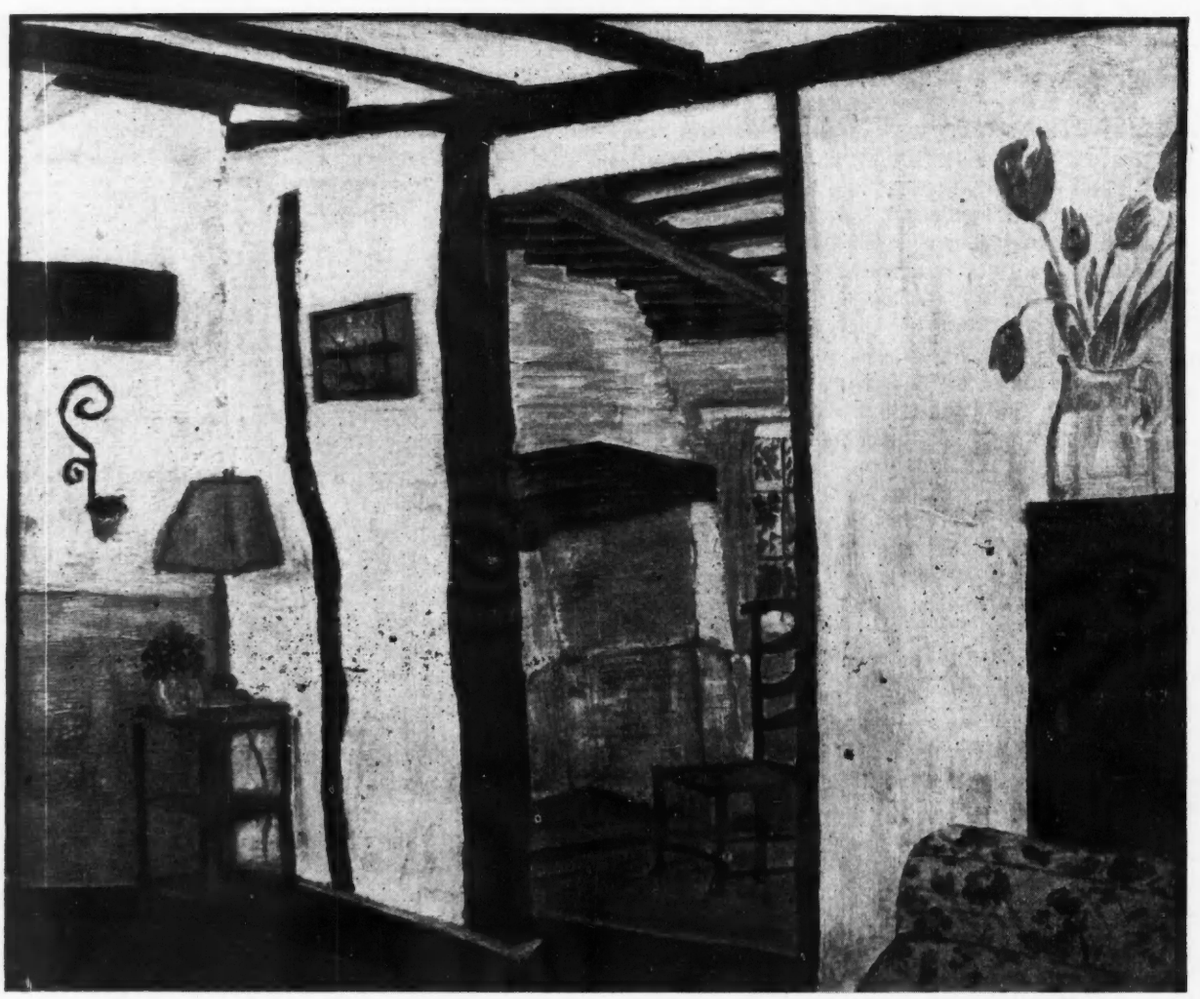
In one of Dr. Gesner's papers he refers to the natural philosopher in the following prophetic words:

"Although he may toil in silence, and remain unknown, and may not receive the least encouragement among his labours, or reward for his pains, yet, when he disappears he leaves something in the hands of his successors that may administer to their wants, and render them wiser and happier."

For seventy years Gesner's grave was unmarked, known only to the caretaker of Camp Hill Cemetery. In 1933, the Imperial Oil Company, erected a fine black granite shaft to "Abraham Gesner, M.D., the inventor of Kerosene Oil". The following is from *Imperial Oil Review*, June, 1933:

"Although the process invented by Dr. Gesner is in use today with certain mechanical improvements, he himself reaped little benefit and until the Curator of the Nova Scotia Museum called attention to his unmarked grave, he has been practically a forgotten man. As a

Fine Art and Camera Salon, Toronto, 1948



"County Cottage". By Dr. Adrian Anglin, Toronto. One of the prize-winning oil paintings.

[Sponsorship of Messrs. Frank W. Horner, Ltd., Montreal]

tribute to his achievements and in gratitude to one whose work has meant so much to one of the world's greatest industries, Imperial Oil Limited has donated a sum of money to be spent on a suitable monument which will be erected to Dr. Gesner.''

In collaboration with the Nova Scotia Historical Society and Nova Scotia Institute of Science the monument was erected in 1933, to a man who started life as a country doctor, sacrificed his material interests for his love of science, experienced the pangs of poverty and shattered hopes, and died poor. Yet, his invention has brought wealth to many and light and comfort to millions of homes.

MEDICAL SOCIETIES

The Canadian Rheumatism Association

The annual meeting of the Canadian Rheumatism Association was held in Toronto on June 22, 1948. The morning session, at Sunnybrook Hospital, opened with a business meeting of the Executive Committee, followed by a discussion of the problem of Ankylosing Spondylitis and a presentation of a variety of clinical cases by the staff of the Arthritis Centre of Sunnybrook Hospital.

Following a luncheon at Sunnybrook Hospital, the afternoon session was held at the Royal York Hotel where the program comprised the following presentations: (1) ''Denervation of Hip Joints'', Dr. R. I. Harris, Toronto; (2) ''Report of Investigation of Hyaluronidase Inhibitors in the Sera of Patients with Rheumatic Disease'', Dr. Hugh Starkey and Dr. Lyon Lapin, Montreal; (3) ''Some Aspects of Rheumatic Treatment in Great Britain'', Dr. H. S. Robinson, Banff, Alberta; (4) ''Some Observations on Nutrition and Protein Metabolism in Rheumatoid Arthritis'', Dr. Victor Schencker and Dr. L. G. Johnson, Montreal; (5) ''Milkman Syndrome'', Dr. J. A. Blais, Montreal; (6) Remarks by Dr. Philip S. Hench, Rochester, Minnesota; (7) Motion Picture by Dr. W. S. Barnhart, Ottawa.

Dr. Philip S. Hench, of the Mayo Clinic, Rochester, Minnesota, was made the first honorary member of the Canadian Rheumatism Association in recognition of his extraordinary contributions to the objects of this organization.

The following officers were elected for the term of 1948-1949: *President*—Dr. Henry P. Wright, Montreal; *First Vice-president*—Dr. A. W. Bagnall, Vancouver; *Second Vice-president*—Dr. W. S. Barnhart, Ottawa; *Secretary-Treasurer*—Dr. Donald C. Graham, Toronto.

CANADIAN ARMED FORCES

News of the Medical Services

The annual meeting of the Defence Medical Association of Canada is being held at the Chateau Laurier, Ottawa, on November 4, 5 and 6, 1948, under the chairmanship of the President, Colonel L. H. Leeson, O.B.E., of Vancouver, B.C. The provincial branches are each sending delegates, but the executive wishes it made known that the meeting is open to all members, and hopes for a large attendance. The annual dinner takes place on Friday evening, November 5.

The Association was founded in 1892 and has since held central meetings in various Canadian cities once and sometimes twice annually except during and immediately following the two World Wars, when most of the members were on active service. Its function is to consider all medical problems affecting the Armed Forces, and it has lent much good counsel and faithful support to the Medical Services. Membership is drawn from medical officers and retired medical officers of the Navy, Army and Air Force.

Dr. J. Paul Laplante, Ste. Anne's Hospital, Ste. Anne de Bellevue, Quebec is the Honorary Secretary-Treasurer.

The first and second year medical cadets of the Canadian Officers Training Corps took part in exercise ''Hippocrates II'' at Meaford, Ont., on August 7, 8 and 9, 1948. The officer cadets and the directing staff from the R.C.A.M.C. School proceeded from Camp Borden to the Meaford Tank Range in convoy on the morning of Saturday, August 7. The first year cadets acted as combatant troops, those of the second year as medical personnel. The use of smoke bombs and blank ammunition gave the scheme a decided air of actuality. The second year students had an opportunity of testing their teaching in the establishment and moving of Regimental Aid Posts and Casualty Collecting Posts. The final siting of the C.C.P. in an abandoned farmhouse was realistic enough to recall wartime scenes to veteran medical officers. Brigadier W. L. Coke, O.B.E., Director General of Medical Services, Canadian Army, was present throughout the exercise, and spoke to the cadets at the conclusion, complimenting them upon their enthusiasm and efficiency.

Four Reserve Force field ambulance commanders arrived at Camp Borden on August 11, 1948, for a three-day visit at the R.C.A.M.C. School. As the School offers courses to members of the Reserve Force as well as to the Active Force and the C.O.T.C. the visitors were particularly interested in observing the training methods and materials which are now in use. The officers were: Lieut.-Col. R. A. Hicks, 8 Fd. Amb., Calgary; Lieut-Col. R. A. Gordon, 7 Fd. Amb., Toronto; Lieut.-Col. C. R. Stephen, 9 Fd. Amb., Montreal and Lieut.-Col. J. H. Shaw, 21 Fd. Amb., Charlottetown.

S./L. E. O. Campbell has been transferred from the Institute of Aviation Medicine, Toronto, as Senior Medical Officer, R.C.A.F. Station, Goose Bay.

SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

INDEPENDENT DOCTORS

Throughout the later stages of the negotiations preceding the introduction of the national health service the veteran Lord Horder became the unofficial leader of the not inconsiderable section of the profession who were opposed to accepting the final compromise. He has now been largely instrumental in setting up an informal committe with a view to sounding the profession as to whether there is any support for the view that a professional organization should be formed for those doctors who do not wish to take part in the new service. In a statement to the press Lord Horder, who is a member of the council of the British Medical Association, has said that ''there is no intention of opposing the British Medical Association, but rather of seeing that the British Medical Association does not in the future give way on any vital points''. What he is anxious about is that, ''should the Ministry of Health turn the screw, economically or otherwise'', there should be

an efficient means of ensuring that the future of the profession is not compromised.

It has been estimated that in England and Wales some 3,000 general practitioners have so far refrained from joining the national health service, but there are some who believe that many more who have actually entered the service would join such an independent organization were it set up.

The Organization of Specialists

The British Medical Association, although often able to speak for the profession as a whole, has always been predominantly a general practitioners' body. Specialists and consultants have often played a prominent part in its deliberations, but they have never looked upon it as an organization to defend their interests. Indeed, until the introduction of the new national health service the specialists of the country pursued an intensely individualistic course so far as conditions of practice were concerned. Their Colleges, outstanding institutions though they undoubtedly were in the national life, devoted most of their energies to such matters as teaching, research and the awarding of higher diplomas. With the advent of the new service it became evident that the specialists and consultants of the country required some co-ordinating body to maintain contact with the Minister of Health. At the annual meeting of the British Medical Association in July it was proposed that the British Medical Association should be responsible for the representation of specialists as well as of general practitioners. This was not to the liking of the bulk of specialists who, not unnaturally, felt that their interests might well have to take second place compared with those of the much larger body of general practitioners.

To overcome these differences a special conference has recently been held between the Royal Colleges and the British Medical Association. As a result of their deliberations it has been recommended that a joint committee of the Royal Colleges, the Royal Scottish Corporations and the British Medical Association should be established to advise the Minister of Health on all matters concerning consultants and specialists. As this committee is to be briefed by both the British Medical Association committee of consultants and a committee organized by the Royal Colleges and Corporations, the compromise is by no means as yet guaranteed of success, but with goodwill on both sides there is every reason to believe that a satisfactory *modus vivendi* will be evolved.

Musical Therapy

An interesting innovation in mental therapy has just been made public. Last November a Council for Music in Hospitals was instituted for providing concerts in hospitals. This followed the initiative of a committee composed largely of medical superintendents of mental hospitals who had experimented with the therapeutic value of music in their hospitals. Since the new Council was formed 65 monthly concerts have been given and there are now 24 hospitals participating in the scheme. So far only mental hospitals have been included. Musicians, both professional and amateur, have been most helpful in taking part in these concerts for very moderate fees. The results have been so promising that a subcommittee has now been set up to investigate methods of research into the rôle of music in the treatment of mental disease. The relationships between the entertainment, cultural and therapeutic aspects of music should provide much food for thought.

A Famous Hospital Ship

Memories of two wars are revived by the announcement that the Bibby liner, *Oxfordshire*, has returned to her home port for "demobilization". Built in 1912, the *Oxfordshire* was the first ship to be requisitioned in the 1914-18 war. As a hospital ship she served in several theatres of war, including Gallipoli and, although she steamed more than 170,000 miles, she was never out of commission. After the cessation of hostilities she returned to her peacetime rôle in the Eastern trade, sailing between the United Kingdom and Ceylon and Burma. The outbreak of war in 1939 again saw her requisitioned as a hospital ship. During the 1939-45 war she spent most of her time as a naval base hospital ship at Freetown, but also served in the Mediterranean and the Far East. Since the end of the war she has repatriated prisoners of war from Hong Kong, as well as serving as a hospital ship in voyages to and from the Far East, the Mediterranean and Germany.

There can be few ships which can emulate this record, and it is good to know that the old campaigner has still a further period of useful service in front of her—this time as an emigrant ship plying between the United Kingdom and the Dominions.

William A. R. Thomson

London, September, 1948.

ABSTRACTS FROM CURRENT LITERATURE

Medicine

Bone and Joint Pain in Leukæmia, Simulating Acute Rheumatic Fever and Subacute Bacterial Endocarditis. Aisner, M. and Hoxie, T. B.: *New England J. Med.*, **238:** 733, 1948.

Examples are described of cases of acute lymphatic leukæmia which simulated rheumatic fever and bacterial endocarditis. The disease can also be mistaken for rheumatoid arthritis. Complete examination of the blood is recommended in all cases with bone and joint pain. X-ray examination will show bone changes in many cases of leukæmia, the most common being periosteal elevation, which is not specific. Other roentgenographic findings may be osteosclerosis, osteoporosis, cortical stratification, rarefaction at the ends of long bones, spontaneous fractures, large tumour masses in the upper ends of the long bones, various osteolytic lesions and medullary striations. Norman S. Skinner

Effect of Surgical Operations on the Bromsulfalein-Retention Test. Tagnon, H. J., Robbins, G. F. and Nichols, M. P.: *New England J. Med.*, **238:** 556, 1948.

Several investigators have reported disturbed liver function in a high proportion of postoperative cases. The authors are critical of some of this work which employed the hippuric acid test as an index of hepatic function, a test which depends upon the integrity of kidney function which is frequently impaired in the postoperative period. Also, trauma to the liver resulting from its being handled during abdominal operations may disturb hepatic function *per se* and not as a specific sequel of surgery.

The authors employed serial estimations of bromsulfalein-retention in 20 cases having operations outside the abdominal area. Normal control tests were secured prior to surgery but an abnormal dye retention occurred in 14 cases during the day or two following operation. There was a certain degree of correlation between the severity of the operation and the degree of dye retention. Other factors, such as type of anæsthesia and age of patient appeared less important. The liver dysfunction was transitory, returning to normal within a few days, and alterations in the cephalin-cholesterol flocculation and thymol-turbidity tests did not develop. These results confirm the work previously done and indicate that definite impairment of liver function is a direct result of protracted surgical operations.

Norman S. Skinner

Evaluation of Curare in Spasticity due to Spinal-Cord Injuries. Kuhn, R. A. and Bickers, D. S.: *New England J. Med.*, **238:** 615, 1948.

Curare has been of clinical value in a number of conditions necessitating muscle relaxation such as tetanus, convulsions incident to shock therapy and the spasms of children with cerebral diplegia. This drug has found its maximal usefulness to date in anæsthesiology. Severe spasm of muscle groups innervated below the level of a spinal cord lesion leads to great difficulties in treatment and may lead to permanent contractures of tendons and joints and prevent ambulation. Bladder management is difficult to achieve in such patients.

The authors have employed a variety of methods in an attempt to overcome muscular spasm resulting from spinal cord injury. The parenteral administration of prostigmine and atropine was not successful. Local injection of spinal nerves with procaine or related preparations gave only transitory relief. Anterior rhizotomy was performed only as a last resort. Thirty-four patients with traumatic lesions of the spinal cord were studied in an attempt to evaluate the use of curare in the treatment of the resultant spasticity. Seventeen patients were given intramuscular injections of 175 mgm. of d-tubocurarine chloride in oil and white wax every 48 hours for 10 doses. In the other 17 intramuscular injections of saline were employed. Beneficial results occurred in both groups but this was due to suggestion since it was impossible for observers to estimate correctly which patients were receiving d-tubocurarine and which were receiving saline. Curare would apparently have no value in the therapy of spasticity resulting from spinal-cord injury. NORMAN S. SKINNER

Food Sensitivity in One Hundred Allergic Children. Hill, L. W.: *New England J. Med.*, **238:** 657, 1948.

Food is much less important as a causative factor of asthma in children than either environmental allergens or upper respiratory infection. A study of 100 asthmatic children showed 35 reacting to one or more foods by scratch tests. Altogether there were 218 positive scratch tests to foods of which 20% were proved to have etiological significance with respect to asthma. No proof was found that food had anything to do with the asthma in the 65 children with negative food tests. The mother of the child usually knew before any testing was carried out whether her child was sensitive to a certain food. While any food may cause asthma, from a practical standpoint not many foods are important. Fish, egg, walnut, peanut and chocolate cover the majority of such cases. Wheat and milk are rarely at fault. NORMAN S. SKINNER

Obstetrics and Gynæcology

Lower Segment Cæsarean Section: A New Head Extractor. Murless, B. C.: *Brit. M. J.*, **1:** 1234, 1948.

A new head extractor for low Cæsarean section is described and illustrated. Other methods of delivering the head are discussed. The technique of using this instrument is described, with the modifications necessary in certain circumstances. Advantages claimed for the extractor are listed. ROSS MITCHELL

Postmaturity. Calkins, L. A.: *Am. J. Obst. & Gyn.*, **56:** 167, 1948.

In the case of the placenta weight and the baby weight, growth is rapid up to about the two hundred and sixtieth day. It is much less rapid from then to the two hundred and eightieth day, and the increment after full term is reached is very small. It is, therefore, important in those patients where the size of the infant may be a complication to recognize the large size early. If the infant is large at full term, its further increment in size thereafter will be so little that it will make no real difference in the course of the subsequent labour. The obvious disadvantages of inducing labour in the presence of an unprepared cervix cannot possibly be justified on the basis of this small increment in the postmature baby.

In this present series of 6,000 cases, there was only one patient whose pregnancy exceeded 316 days, and the vast majority of the "postmature" group were delivered at the end of 305 days or less. Thus the problem is one of two or three weeks postmaturity, not one of two or three months. In this two or three weeks the amount of increased ossification is certainly negligible so far as creating a problem for the average patient. ROSS MITCHELL

Serum Beta-Glucuronidase Levels During Toxæmia of Pregnancy. Odell, L. T. and McDonald, D. F.: *Am. J. Obst. & Gyn.*, **56:** 74, 1948.

Serum beta-glucuronidase levels will differentiate most cases of presumable pre-eclampsia and hypertensive toxæmia of pregnancy. Glucuronidase levels may warn the obstetrician of an impending pre-eclampsia. The serum concentration of the enzyme does not reflect the severity of the toxæmia. Levels during convulsive toxæmia are not diagnostic. The serum concentration of beta-glucuronidase apparently reaches a maximum just before, or early in the course of, pre-eclampsia, and then falls during the duration of the syndrome. ROSS MITCHELL

The Use of Post-Pituitary Extract in Physiological Amounts in Obstetrics. Theobald, G. W., Graham, A., Campbell, J., Gange, P. D. and Driscoll, W. J.: *Am. J. Obst. & Gyn.*, **56:** 88, 1948.

A dilution of the oxytocic principle of post-pituitary extract in the blood plasma of an order not exceeding $1:375 \times 10^6$ is capable both of initiating and of augmenting labour pains in man. This dilution is comparable to that of the antidiuretic principle which suffices to inhibit water diuresis. This evidence makes it increasingly difficult to believe that the post-pituitary gland is normally concerned either with regulating or with elevating the blood pressure.

The use of pituitrin-quinine drip is of great value in those cases in which the woman fails to go into labour subsequent to the rupture of the membranes, whether spontaneously or as the result of surgical intervention. The pituitrin drip increases both the frequency and the intensity of the uterine pains in cases of uterine inertia and makes possible the adequate use of sedative drugs in the conduct of labour. The pituitrin drip may be used in all cases in which it is considered desirable and safe to stimulate the uterine pains, and it is immaterial whether the woman is a primigravida or a multigravida whether or not she suffers from hypertension, or whether the head is above the brim. It is not impossible that it will be found advantageous to apply the pituitrin drip to a wider range of cases. It is also possible that it will find a place in the third stage of labour, particularly in those cases of severe post-partum hæmorrhage in which the placenta is retained. The pituitrin would of course be added to the transfused blood.

Pitocin would appear to be as efficacious as pituitrin and it would perhaps be more logical to use pitocin particularly in those cases which manifest hypertension. ROSS MITCHELL

Intracranial Hæmorrhage in the Newborn. Saunders, C.: *J. Obst. & Gyn. Brit. Emp.*, **55:** 55, 1948.

The diagnosis of intracranial hæmorrhage is rendered difficult by the fact that the examination is purely objective, and clinical methods of approach have little value where the central nervous system of the newborn is under consideration. The tendon reflexes are inconsistent and often misleading. Examination of the retina is difficult, and in addition has been reported to show hæmorrhage as a result of congestion in 20 to 50% of all deliveries, according to different observers.

The pyramidal tracts are non-medullated, and presumably not functioning, which rules out the forebrain as an aid to localizing the trouble. The fontanelle may be bulging in cases of gross hæmorrhage or in cerebral œdema, but may appear completely normal where the hæmorrhage is small or localized infratentorially. It is a sound policy to treat every case of asphyxia as an intracranial hæmorrhage until proved to be otherwise. The birth history is often of great assistance in making a diagnosis. The infant may show coma or drowsiness and refuse to suck, or may be extremely restless and irritable with a "cephalic" cry, and a tense, terrified expression. Vomiting for no apparent reason is sometimes a symptom. A slow pulse and rapid, shallow breathing, combined sometimes with thermolability, lethargy, coma or cyanotic attacks may be associated, with hæmorrhages involving the medullary area.

P. J. KEARNS

Acute Rheumatism in Pregnancy. McKeown, F.: *J. Obst. & Gyn. Brit. Emp.*, **55:** 50, 1948.

Rheumatic heart disease, even in its severest form, may occur subclinically. Recrudescences of rheumatic fever during pregnancy in a patient with a known cardiac lesion may not infrequently be the factor responsible for cardiac defeat. The patient with active rheumatic carditis may die quite suddenly following delivery, the condition simulating acute obstetric shock.

P. J. KEARNS

A Benign Tumour of the Placenta. Davies, D. V.: *J. Obst. & Gyn. Brit. Emp.*, **55:** 44, 1948.

A hæmangioblastoma of the placenta is described. The view is advanced that these arise early, from angioblastic strands which take on unrestricted growth and may or may not completely lose their continuity with the primitive angioblastic network, or, having lost this continuity, may fail in part or wholly to re-establish it.

P. J. KEARNS

Gross Hypertrophy of the Pregnant Uterine Cervix Simulating Cancer. Hill, A.: *J. Obst. & Gyn. Brit. Emp.*, **55:** 31, 1948.

A case is described of a multigravida, aged 26 years, in whom gross hypertrophy of the uterine cervix with blood-stained discharge led to an erroneous diagnosis of cancer in the 37th week of pregnancy. Immediate Cæsarean section produced a healthy child and rapid healing of the cervix following a puerperal course of sulfathiazole was noted. Equivocal biopsy-findings are recorded and the facts regarding cervical cancer in pregnancy are summarized.

P. J. KEARNS

Arachnodactylia. Dorrance, T. O.: *J. Pædiatrics*, **31:** 679, 1947.

Arachnodactylia, or Marfan's syndrome, is characterized by a congenital symmetrical malformation of the extremities consisting of considerable elongation and attenuation of the bones, more pronounced in the distal parts. The etiology is not known, however; one theory is that it is a fault in the mesodermal cells of the embryo. One-third of the reported cases are hereditary and familial.

Clinically, the syndrome seems to be allied to amyotonia congenita. There is marked atrophy of the entire musculature, especially the extremities. The subcutaneous fat is diminished in amount. The bones of the hands and feet are increased in length. Abnormalities of the skull, usually towards dolichocephaly, are present in about 80% of cases. Bilateral abnormalities of the eyes occur in over half of the cases. Luxation of subluxation of the lens is the most frequent abnormality. Congenital heart disease has been noted in about a third of the cases. There is no satisfactory treatment. There is a low resistance to respiratory infection and, as a result, few of the children reach maturity. A case is presented with the typical features.

PRESTON ROBB

Pædiatrics

Reablement of Children with Infantile Cerebral Palsy. Collis, E.: *The Lancet*, **253:** 239, 1947.

Cerebral palsy is a dysfunction of movement due to a cerebral lesion interfering with one or more of the several pathways directly concerned with normal human movements. The outstanding characteristic of infantile cerebral palsy is imperfect motor control due to cerebral damage inflicted on a growing organism. The appearance and behaviour of these patients often lead to an inaccurate diagnosis and subsequent mistreatment of the child until he fits his diagnosis and becomes mentally and physically incompetent. Damage may, however, be so slight that it is undetected and motor disability passes for clumsiness, laziness or mild mental defect where the handicap is hidden, *e.g.*, athetosis of the extra-ocular muscles or partial deafness. It is important in handling these cases to classify them properly. They have been divided into 5 types, spastic, athetoid, ataxic, rigid and tremor. At the Queen Mary's Hospital for Children the child is admitted for a probationary period into a treatment unit to allow proper classification and conditioning to his environment. Assessment of the total handicap, as far as present permits, is made as follows: (1) Functional ability. (2) Nutrition and appearance. (3) Etiology. (4) Classification into any one of the 5 motor groups already named. (5) Analysis of specific disability. (6) The dominant handedness, and (7) the mentality. As soon as the patient is properly classified training is instituted as follows, all measures overlapping to form a whole rehabilitation scheme. (1) Physiotherapy; (2) every day activities; (3) occupational therapy; (4) speech therapy; and (5), school or kindergarten work. A rounded program is worked out which covers all aspects of the patient's need. The author points out the necessity for early care by people properly trained in this field. They feel that contrary to some opinion training of these patients is very worthwhile. Physiotherapy is based on the concept that in spasticity the lesion is pyramidal whereas in athetosis the lesion is extrapyramidal; the damage to the cortex leads to a type of motor dysfunction which differs essentially from that due to a basal damage. It was felt that dural lesions are actually rare though commonly children are thought to have a dural lesion and that tension athetosis is commonly diagnosed as spasticity. Mechanical aids are introduced only to be discarded as progress is made. These include skiis for walking, individually adapted chairs and tables, wooden-handled spoons and so on.

PRESTON ROBB

Nature and Treatment of Stuttering. Glasner, P. J.: *Am. J. Dis. Child.*, **74:** 218, 1947.

Stuttering has its essential origin in early childhood, usually between the ages of 2 and 4 and is a manifestation of some emotional disturbance. Unless the physician becomes familiar with the causes and nature of stuttering, it will be impossible to reduce the incidence of this condition which afflicts approximately 1% of the population. Contrary to earlier beliefs, it has been found that the causes of stuttering are of a functional or psychogenic nature rather than of physical origin. Any condition or situation which can produce disturbances of a behavioral or emotional nature in children may precipitate stuttering. As a rule, the causative factors are multiple. Conditions which create feelings of unrest, insecurity or tension may bring on the condition.

Some of the factors are: (1) Overstimulation or pampering, particularly in homes where more than two adults are present. (2) Overconcern about eating, on the part of the parents. (3) Inhibition of the freedom and relaxation essential to the child's well-being, by an overprotective mother. (4) Inconsistency of treatment by the mother and by the father. (5) Overconcern over

the normal repetition of children by parents who stuttered or had relatives who stuttered. (6) Feelings of insecurity based on quarreling between parents and fear of pending separation. (7) Insecurity based on frequent changes of residence. (8) Confusion and tension on part of child due to overfrequent correction by parents or errors of articulation, pronunciation or grammatical construction. (9) Feelings of insecurity produced in an older child by improper handling after the birth of a sibling. (10) Sibling rivalry.

Stuttering, unlike many other disorders of childhood, becomes progressively worse as time goes on if the causes are not removed. Sooner or later there develops a feeling of expectancy to stuttering and he will invariably stutter again in these situations. He seems to acquire a "conditioned response". Treatment should be based on the results of careful examination, which takes into consideration such factors as causes, age, personality, present environment, attitudes, emotional problems and, of course, symptoms. Treatment should be individual and one should treat the individual as a whole. Since stuttering can have such far-reaching effects on the emotional, social, educational and vocational life of a person, it is imperative that treatment be initiated as soon as it is indicated. PRESTON ROBB

Industrial Medicine

Communication Between People in Industry. Radcliffe, R. A. C.: *Indust. Welfare & Personnel Management*, **30:** 46, 1948.

One of the most important and difficult problems in industrial management today is the improvement of management-employee relationships. The author of this article presents a broad outline of the problem as a whole and draws attention to the various means by which mutual understanding, with full co-operation between management and men, can be arrived at. It is his contention that men must be given not only an understanding of what is being done, but a real feeling that their point of view and their interests really matter. Under the headings of verbal, written and visual methods of communication, he discusses the various ways by which the problem can be solved. The advantages and limitations of each method are considered together with ways of combining the methods. Certain general rules applicable to all three methods include the following: (1) The meaning must be expressed in a way which can be understood by everyone and in a way which will evoke the right response. (2) Management must have a clear knowledge of what the workers want to know, and what they, *i.e.*, the management, wish to tell them. (3) Too many ideas should not be presented at once.

The main methods of verbal communication discussed are: informal talk, interview, speech, committee meetings and discussion groups. The great advantage of verbal methods over written, is the personal contact established between speaker and audience.

In the author's opinion written communications are difficult to do well; the best written ones can rarely be as effective as good verbal ones. The principle methods are: employee handbooks, works magazines or bulletins, notices, suggestion schemes and annual reports and accounts. The rôle of each is discussed. In all, simple words which have no double meanings, and short sentences, are essential. Wherever possible, visual aids should be used to illustrate both verbal and written communications. The most important are posters, pictures, films and exhibitions. The author stresses the importance of every firm constantly learning through experiment and observation, any new techniques which are developing. Much skill and knowledge is required to balance the right method of communication with the right manner, the right moment and the right audience.

MARGARET H. WILTON

Allergic Problems of the Railway Surgeon. Feinberg, S. M.: *Indust. Med.*, **17:** 91, 1948.

In this article the author presents the problem of allergy as it concerns the railway surgeon and indicates the necessity for him to apply the principles of allergy in his practice. After a brief review of allergic manifestations which constitute major problems, specific substances responsible for allergic reactions, diagnosis of allergy, and principles of treatment, the allergic problems within the sphere of the responsibilities of the railway surgeon are discussed as they fall into three main categories. These categories are: (1) non-occupational allergy among employees; (2) occupational allergy; (3) allergy relating to passengers and public.

As 5 to 10% of the population are allergic, allergy not due to occupational factors, among railway employees, is quite common. These allergic ailments are important as they may affect efficiency and alertness. They must be considered in the pre-employment examination, particularly in determining the job classification. Furthermore the asthma or hay fever of an employee may necessitate changing his job from outdoors to indoors, or from railway station to city office. A number of specific allergens are encountered in the railway industry. Among occupational inhalants causing allergy are flour and grain dust in freight handlers, and upholstery dust in porters. Such allergens as pollen and mould spores, although not truly occupational, present a definite problem to the outdoor railway worker. A freight handler may encounter many substances that produce contact dermatitis. The importance of coal smoke is stressed. To any employee with a known respiratory allergy, the inhalation of coal smoke will present a serious hazard. Such persons should avoid jobs which expose them intimately to such atmospheres. From the standpoint of the public also, coal smoke is the most prolific source of annoyance among allergic individuals. The opinion is expressed that a great deal of the asthma and rhinitis which people experience in the city is due to the coal smoke from the railway and other industries.

The large number of allergic travellers constitutes a large-scale travel problem. In this connection the author makes certain suggestions regarding conditions on trains. For those persons allergic to dust and feathers, the provision of two or three sets of mattresses and pillows enclosed in dust-proof casings would provide comfort; for individuals allergic to certain foods, simple modifications of diets, particularly wheat, egg, and milk-free diets, could be offered. Such innovations would gain deserving publicity for those railway lines providing them.

MARGARET H. WILTON

OBITUARIES

Dr. Matthew George Burris died in Dartmouth, N.S., on August 18. He was 61 years of age. Born at Upper Musquodoboit, February 28, 1887, he attended Truro Academy, continuing his studies at Dalhousie University, where he obtained his B.A. degree in 1907. He graduated in medicine from Dalhousie in 1911. Upon graduation, he practised for a short time at Kamloops, B.C., returning east in 1912 to establish his practice in Dartmouth.

A valued member of the congregation of St. James United Church, Dr. Burris had an intense interest in town affairs, was a charter member of the Dartmouth Memorial Hospital Fund, and he served on its Board of Directors since the fund's inception. Keenly interested in sports, he was a member of the Brightwood Golf Club. During his college days he was star member and team captain of the glorious Dalhousie senior rugby team of 1909, which was one of the greatest teams in the history of the University.

A member of the Nova Scotia Historical Society, he travelled extensively in the province, taking a keen

interest in its history, writing many short stories based on Nova Scotia lore. He was a former president of the Halifax Medical Society, a member of the Nova Scotia Medical Society and of the Canadian Medical Association. He is survived by his widow, one daughter, two brothers and four sisters.

Dr. Manuel Gorin de Souza, physician and surgeon, died August [illegible] at his home in Toronto. Dr. de Souza had practised in Toronto for 23 years. He was born in British Guiana and graduated from the University of Toronto in 1924. He took post graduate work in New York. He was a member of St. Anthony's church and the Catholic Order of Foresters.

He is survived by his widow and two sons.

Dr. Edward J. Finnerty, aged 59, former resident of Toronto, died July 14, in Sonora, Calif. Born in Toronto, he attended St. Michael's college where he graduated in medicine in 1911. He practised at Henley, Sask., for 15 years, and moved to southern California where he was chief of staff at Eldridge state hospital. He is survived by his widow, two daughters and a son.

Dr. Mervyl Homer Winifred Fizzell, aged 61, staff member of the Colonel Belcher Hospital, Calgary, died recently at his home of a heart attack. Graduate of Queen's University, he practised in Saskatchewan before going to Calgary in 1942. A member of the Masonic Order and the I.O.O.F., he is survived by his widow, a daughter, his mother, two sisters and two brothers.

Dr. C. E. Fortin, died in Santa Monica, California, on August 9 following a lengthy illness. He graduated from the University of Manitoba and McGill University, doing postgraduate work in London, England. He served overseas with the R.C.M.C. during the First World War. At various times he practised in Newfoundland and Winnipeg. He is survived by his widow, a son, a daughter, a brother and a sister.

Dr. W. Leighton Gilbert, aged 68, medical practitioner in Toronto for many years, died August 24. He was born in Picton and was a graduate of the University of Toronto. He is survived by his widow.

Le Dr F.-X.-P. Goyette, autrefois de Sorel, est décédé le 29 juillet à sa résidence à Montréal. Il avait fait ses études au collège de l'Assomption et reçu son doctorat à l'Université de Montréal. Il avait pratiqué sa profession pendant 54 ans. Il était l'ami des pauvres et avait été pendant 25 ans président de la S.-Vincent-de-Paul, paroisse S.-Edouard. Il laisse son épouse et trois sœurs.

Dr. A. W. Haldimand died on July 31, in Montreal, in his 86th year. Born in Montreal, he graduated from McGill in 1886. For many years he was medical director of the Thomas Davidson Co., now the General Steel Works, *The Montreal Daily Star,* and the Wire and Cable Co. He retired from active practice in 1937 after serving 20 years as medical director to the Northern Electric Co. He was formerly an active member of the Royal Montreal Curling Club. He is survived by his widow and two sons.

Dr. Ashton S. Langrill died on August 21 at Christie St. Hospital, Toronto. Born in Jarvis, he was a graduate of the University of Toronto in 1894. Dr. Langrill practised at Atkwood and in Hamilton just prior to the outbreak of the First Great War. He served in France with the Royal Canadian Army Medical Corps and was with the Haldimand Rifles, Canadian Militia, for 25 years with the rank of Lieut.-Col. He is survived by his brother and two sisters.

Dr. John Edgar McLean, aged 65, died at St. Joseph's Hospital in Sudbury on July 23. A native of Orillia, Ont., he was medical officer of health for the town of Capreol, and one of the pioneer medical men of the Sudbury district. He is survived by his wife, a son, four daughters and a brother.

Dr. James Archibald MacLellan, aged 81, retired medical specialist, died at his home in Sydney, N.S., on July 28. Dr. MacLellan was a native of Economy, and after graduating from Queen's University and practising his profession for a period in Economy and Great Village took a course specializing in eye, ear, nose and throat surgery from the New York Infirmary. He came to Sydney in 1910 and set up a practice from which he retired in 1938. Surviving are three sons, a daughter and two brothers. Dr. MacLellan was a member of the Sydney Curling Club, the Royal Cape Breton Yacht Club and the Lingan Country Club and a veteran member of the Cape Breton Medical Society.

Dr. William Morrison, died at Southampton, Ont., on August 12. He was 88 years of age. Dr. Morrison was one of Sudbury's early pioneer general practitioners and he was formerly in charge of the Copper Cliff Hospital for Inco. He was a native of Kirkton Parish of Forghen Banffshire, Scotland. He attended parish school at Black Hills, Scotland and academy at Peterhead, before coming to Canada in 1869 with his parents. He attended public school and Hamilton Collegiate and later taught school for five years in Huron County. He graduated from the University of Toronto, School of Medicine in 1890 and practised in Durham, Pinkerton and Paisley. In 1906 he received his degree as member of the Royal College of Surgeons of England, and became a Licentiate of the Royal College of Physicians of London the same year. He returned to Canada the following year, and practised in Paisley before going to Copper Cliff in 1906. He remained in practice there till 1912 when he took over a private practice in Sudbury. Dr. Morrison was a member of the staff of St. Joseph's Hospital, Sudbury, from 1912 till 1941. In 1941 he retired, moving to Southampton. He was jail doctor in Sudbury for many years and also served in lumber and mining camps of that district. Surviving are his widow, two sons, a brother and a sister.

Dr. Robert L. Murray, a native of Cape Breton died at the home of his daughter, Mrs. Kenneth Spencer, Moncton, N.B., on July 15. Born at The Narrows, Cape Breton, in 1860, Dr. Murray practised medicine in Pictou and Springhill, later becoming assistant superintendent of the Nova Scotia Hospital, Dartmouth. He retired in 1927 and lived at the North Sydney family home, coming to Moncton three years ago. He is survived by his widow, one son and two daughters.

Le Dr J.-Aldéric Saint-Denis est décédé recemment à Montréal. Il était âgé de 77 ans. Né à Vaudreuil en 1871, il avait fait ses études au collège Bourget de Rigaud et à l'Université Laval de Montréal. Reçu médecin en 1894, il avait exercé sa profession d'abord dans son village, puis était venu s'établir à Montréal. Par la suite, il avait poursuivi ses études à Paris, s'était spécialisé dans le traitement des maladies des yeux, de la gorge et des oreilles. Il s'était retiré en 1943. Outre son épouse, il laisse deux filles, deux frères, et trois sœurs.

Dr. Moses Scherzer, prominent Montreal pædiatrician and actively associated with the Jewish General Hospital and the Herbert Reddy Memorial Hospital, died suddenly on August 12, at Old Orchard Beach, Me., where he was holidaying with his family. He was in his 50th year. Honorary president of the Jewish Child Welfare Bureau, Dr. Scherzer was a director of the Baron de Hirsch Institute, president of the Montreal Clinical Society and a former member of the American Pædiatrics Society. Active in Red Cross work during the late war, he was also on the staff of

the Mount Sinai Sanatorium at Ste. Agathe. He was a member of the Montefiore Club, St. George's Lodge No. 10, A.F. and A.M. and an alumnus of Pi Lambda Phi fraternity. A native of New York City, Dr. Scherzer moved to this city at an early age, attending Montreal High School and graduating from McGill University in 1918. In 1923 he did postgraduate work in Berlin and Leipzig.

Surviving are his widow, a son, a daughter, his mother, and a brother.

Dr. Alfred Brodie Stewart, who after fifty years of practice at Plumas, retired only last November, died on August 5 at Binscarth in the home of one of his three daughters. He graduated in medicine from Manitoba Medical College in 1895 and in 1933 was made a Life Member of the College of Physicians and Surgeons of Manitoba.

Dr. Frank A. Sullivan, aged 68, died suddenly of a heart attack on July 19, in Halifax. Born in St. Stephen he graduated from the Tufts Medical School, Boston, and was house physician for many years at the Palliser Hotel in Calgary, retiring in September, 1947, when he returned to the Maritimes and made his home in St. Stephen and Halifax. He is survived by three sisters, and three brothers.

Dr. H. F. Tyerman died on August 18 in the Royal Inland Hospital at Kamloops. He was born and raised on a farm 65 years ago at Seaforth, Ont. He graduated from the Toronto University in 1906 and immediately went to Kindersley, Sask., where he practised during the horse and buggy days and had many trying experiences during the cold winters fighting the blizzards. In 1928, he came to British Columbia on account of a chest condition he had developed and settled at Nakusp on the Kootenay Lake; while there he established a splendid reputation for practical work and good surgery. In 1942, he went to Ashcroft where he looked after a large area in the Cariboo. The work was heavy for him but he stuck to it. During the past year he was in bad health and had to bear with much suffering. He leaves his widow and his son, Don Tyerman of Vancouver.

NEWS ITEMS

Alberta

Dr. L. J. Patterson of Red Deer has recently been attending clinics at the University of Alberta hospital.

The epidemic of poliomyelitis appears to be subsiding throughout the province. Like other parts of Canada the disease was not concentrated in any centre but sporadic throughout with few deaths fortunately.

Dr. Gordon Bell has returned from London, England, where he was doing special cardiac investigation and cardiology. He has resumed his practice with his father Dr. Irving Bell of Edmonton.

Dr. F. H. Mewburn has been made honorary professor (clinical) of Orthopædics at the University of Alberta upon his recent retirement from the faculty, Dr. Mewburn will continue his private practice in the city of Edmonton.

Upon the retirement of Dr. A. F. Anderson as Superintendent of the Royal Alexandra Hospital for twenty years, a banquet was given in his honour at which some 90 members of the profession attended to pay Dr. Anderson honour. Dr. Donald Easton, formerly assistant superintendent now becomes the superintendent of the Royal Alexandra.

The new library of the University of Alberta is well under way and will be a great asset to the rapidly growing University; it is situated East of the medical building. The gymnasium has been moved one block South of its original position and will be close to the University rink. The new Aberhart Memorial hospital for tuberculosis is under construction in the Southern portion of the University grounds.

Dr. J. J. Ower has retired as Dean of the Faculty of Medicine at the University of Alberta. Dr. John Scott has been appointed to fill that position and will be assisted by Dr. H. E. Rawlinson of the Department of Anatomy. We are pleased to learn that Dr. Ower will continue with the University and will direct the pathological and clinical laboratories of the institution.

An error was made recently in stating that Dr. Singleton formerly of Calgary and now at the Coast had died. It so happens that he went to the B.C. coast and is well and hearty in his retirement. Our sincere apologies to Dr. Singleton. W. CARLETON WHITESIDE

British Columbia

The British Columbia Government has now sent out forms to every householder and self-supporting citizen in the Province, which must be filled out and returned, in order that the new Hospitalization Act may be put into force by January 1, 1949. At least six months' dues must be paid or arranged for in advance.

As a result of the new Hospitalization Plan, a great many organizations which provided hospital insurance have had to suspend this branch of their activities, as the coverage that they could provide would not be compatible with that provided by the new plan. Chief of these is the Blue Cross, which ceases its work in B.C. at the end of 1948.

The Government has taken cognizance of the almost certain prospect of a considerable increase in the demand for hospital beds in the near future. An item in the press reports a plan to go ahead with the extension of hospital accommodation, by an addition to the Vancouver General Hospital of some 825 beds. An expenditure of ten to eleven million dollars is contemplated. This extension will be available for the purposes of the medical school shortly to be inaugurated at the University of British Columbia.

It is to be hoped that due attention will be paid to the needs of other cities and towns in British Columbia where the urgency is quite as great, in proportion, as in Vancouver.

The annual meeting of the British Columbia Medical Association, to be held in Vancouver at the end of September, promises to be an extremely important meeting. The clinical part of the meeting includes a program of addresses, clinics, etc., which will be of a very high order. At, or about the same time, several other important meetings are being held. Thus, during the week preceding the actual meeting, the Pædiatric Staff of the Victoria General Hospital is putting on a refresher course for general practitioners. The program includes pædiatricians from all parts of the city, and there will be clinics and demonstrations. It is an excellent program and will undoubtedly attract a great many men. The British Columbia Surgical Society, the British Columbia Internists' Society and some other organizations are also holding their annual meetings at about this time.

A great deal of emphasis will be placed on economic matters at this annual meeting. This is only to be

expected, in view of the developments along lines of socialized medicine in this Province and elsewhere in Canada. One or two announcements of great importance are expected, and the Committee on Economics of the British Columbia Medical Association, has done a tremendous amount of work in these matters.

It is expected that Mr. Pearson, the Provincial Minister of Health, will be present at the meeting, and will address us.

We regret greatly to report the recent death of Dr. George A. Kidd of Vancouver. Dr. Kidd was formerly Professor of Anatomy at Queen's University, and had practised in Vancouver for many years. He was the historian of the Vancouver Medical Association, and had been chronicling its doings for a long time—his writings appearing in the *Bulletin* from time to time.

Dr. W. Ronald Taylor, Vancouver, B.C., has been made a diplomate of the American Board of Otolaryngology at the recent examinations in Chicago.

Plans for an Academy of Medicine for Vancouver are going ahead and Dr. L. H. Appleby of that city is in charge of the campaign to head up these plans, and organize a plan of financing the building by whatever methods may be seen to be best. He is an admirable choice for the position. J. H. MACDERMOT

Manitoba

Dr. Leon Rubin of Rivers has been appointed a coroner in and for the Province of Manitoba.

Professor John McKelvey, head of the Department of Obstetrics and Gynæcology, University of Minnesota, will be guest speaker at the annual meeting of the Manitoba Division of the Canadian Medical Association in Winnipeg, October 19, 20 and 21.

Dr. Steinn O. Thompson of Riverton, M.L.A. for Gimli, was honoured at a large gathering of his friends and patients at Hnausa Park when he was presented with a new Ford car and a grandfather clock. Since the occasion of the gathering was the 25th anniversary of his marriage, and funds more than sufficient to cover the cost of the two gifts, it was decided to add a refrigerator. Characteristically, Dr. Thompson stated that he would accept the car but would set aside the cost as a trust fund for the erection of a hospital at Riverton. Born 55 years ago he served in World War I, graduated in medicine in 1921, and since then has practised on the west side of Lake Winnipeg. His son is a medical student; one daughter is engaged in social work in Winnipeg, and the other is a nurse in training in the Winnipeg General Hospital.

The by-law for the establishemnt of Minnedosa Hospital District No. 8 was passed with a 96% majority. The vote authorizes establishment of the hospital district and the erection of a $130,000 modern hospital containing 30 beds, full operating and diagnostic facilities, office space for doctors and dentists and a maternity wing containing a nursery.

On August 8, the new Boissevain Memorial Hospital was dedicated as a tribute to the sacrifice of Boissevain's war dead. The new hospital will serve the municipalities of Boissevain, Morton and Minto and the village of Minto. Over 125 Canadian Legion members paraded to the hospital grounds to witness the unveiling of a bronze plaque and the dedication by Reeve A. A. Paterson of Morton. Subscriptions totalling over $50,000 were collected for the hospital.

G. E. Delory, Ph.D. with his wife and family have come to reside in Winnipeg where Dr. Delory will be Assistant Professor of Biochemistry in the Faculty of Medicine, University of Manitoba. He was formerly biochemist in the Department of Pathology.

ROSS MITCHELL

Nova Scotia

Dr. Lawrence Sutherland, son of Dr. R. H. Sutherland of Pictou, has accepted a temporary medical appointment in Newfoundland. Graduating this spring from Dalhousie University he is the fourth generation of Sutherlands to practice in Nova Scotia.

The forthcoming Annual Meeting of the Medical Society of Nova Scotia which will be shortly held at Celtic Lodge, Cape Breton Highlands National Park, will be fraught with important issues to the profession in Nova Scotia. A scheme of health insurance will be explored.

At a recent convention of the United Mine Workers of America, District 26, held at Truro, a resolution was passed endorsing co-operative medical schemes for mining areas. At present this is a live issue in the Cape Breton Coal Field, particularly in Glace Bay and New Waterford. H. L. SCAMMELL

New Brunswick

Dr. P. C. LaPorte, of Edmundston, has received fresh recognition of his ability as a skilled wood carver. During the present summer he has exhibited some of his recent work at several American National Conventions, where it attracted much complimentary attention. One notable carving depicts in relief a group of the chief early workers in the production of penicillin.

Dr. Stephen D. Clark was elected to the N.B. Legislature to represent Saint John County at the recent provincial election. Re-elected at the same time were Hon. Dr. F. A. McGrand and Dr. E. R. Kennedy.

A small number of cases of typhoid has been reported in the neighbourhood of Moncton, traceable to untreated water supplies. The situation appears now under control. Only one case of polio has so far been reported in the Province this summer and it was of a mild type.

The general health of the population in New Brunswick must be good this year as more holidays are being enjoyed by more doctors than in any year since 1938. As is usual many of these holiday trips are being used as opportunities for study at clinical centres in Canada and the United States. In such cases a change is better than a rest due to the added knowledge that the holidaying doctor brings back to his community.

Several doctors from Great Britain and Ireland are serving in New Brunswick Hospitals until they write the examinations of the Medical Council of Canada. These well qualified young physicians from the old country are a welcome addition to our Canadian profession.

The following physicians have recently registered with the Medical Council of New Brunswick and are practising at the addresses given: Dr. John Metcalfe, Calais, Me.; Dr. Phil d'Outremont, Moncton, N.B.; Dr. A. A. McVicar, Port Elgin, N.B.; Dr. Denis Stein, St. Quentin, N.B.; Dr. Norman J. Belleveau, Moncton, N.B.; Dr. Lloyd Allen, Hatfield Point, N.B.; Dr. L. H. Freeman, Saint John, N.B.; Dr. Earl R. M. Lee, Black's Harbour, N.B.; Dr. Marcus A. Deacon, Shediac, N.B.; Dr. Gordon L. Mitton, Richibucto, N.B.; Dr. Chas. W. Depow, Canterbury, N.B.; Dr. Peter G. Lyons, Moncton, N.B.; Dr. Melvin J. Acker, St. Stephen, N.B.

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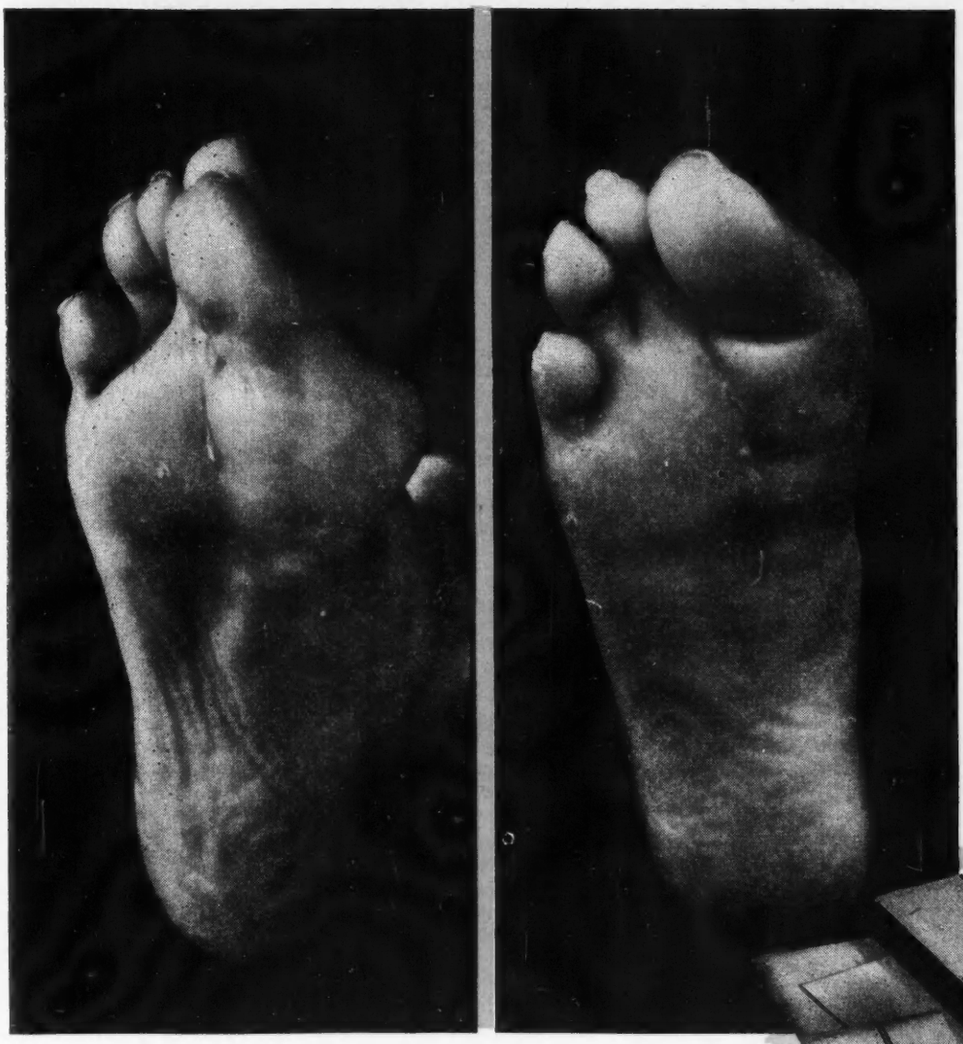

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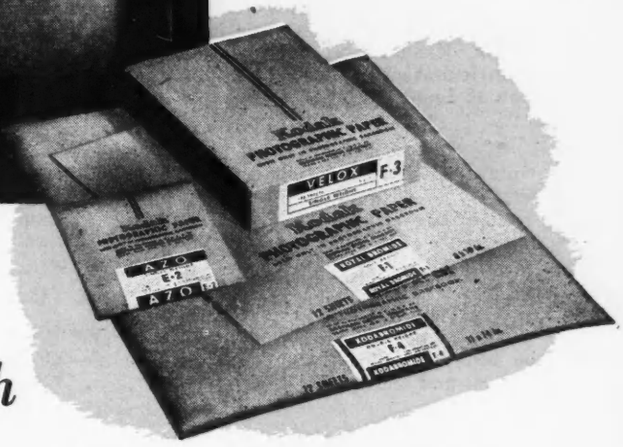

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Ontario

The University of Western Ontario is holding a graduate Lectureship in London, October 12 and 13. Dr. John McMichael, Professor of Medicine at the British Postgraduate Medical School, University of London, will speak on both days. His subjects will be "The Pharmacology of the Failing Human Heart" and "Pulmonary Heart Disease".

District Number Four of the Ontario Medical Association is holding its annual meeting in conjunction with the Clinical Day of the Hamilton Academy of Medicine in Hamilton on October 13. The business session will take place on the evening of the 12th. Twenty-seven local physicians will give clinics and lectures. In the evening a dinner will be held at the Collins Hotel, Dundas, when the organizational meeting of the Section on General Practice of the Ontario Medical Association will take place.

The annual business meeting of District Number Eleven of the Ontario Medical Association (Toronto District) will be held on the afternoon of October 27th at the Academy of Medicine Building. The scientific session will be held in conjunction with the Section on Surgery of the Academy at the Ontario College of Education Auditorium on the evening of November 2. The guest speaker will be Sir Archibald McIndoe, Chief of the Plastic Surgery Centre, Queen Victoria Hospital, East Grinstead, Sussex, England.

At the annual meeting of District Number Two of the Ontario Medical Association in Galt on November 3, Dr. Kenneth N. Campbell of Detroit will discuss "Various Aspects of Renal Failure" and "The Utilization of Lumbar Sympathectomy in Arteriosclerosis".

The annual meeting of District Number Nine of the Ontario Medical Association will be held in Timmins on October 3, 4, and 5. The guest speakers will be Dr. Melville C. Watson, Dr. Alan Brown, Dr. William Boyd of Toronto and Dr. G. S. Williamson of Ottawa. The Honourable Paul Martin will be the after dinner speaker.

The annual meeting of Districts Six and Eight will be held in Peterborough and Renfrew on October 19 and 20 and October 6 respectively.

Lady Banting has returned from England where she interned for two years in Manchester. She was successful in passing the examinations for membership in the Royal College of Obstetrics and Gynæcology.

Dr. Hans Friede has announced the opening of an office in Toronto for the practice of Neurology and Psychiatry. He graduated from the University of Berlin in 1930, then he studied in Leipzig, Konigsberg and Magdeburg. He spent six months working with Dr. C. G. Yung in Zurich, Switzerland. After coming to Canada in 1939, he interned at the Ontario Hospital, Whitby. He now holds a specialist certificate in Neurology and Psychiatry.

Dr. Florence Haslam has arrived at Maple Leaf Hospital, Kangra, East Punjab, India after a year's furlough when she visited her parents in Toronto and did postgraduate work in surgery in Regina.

A membership of 1,100 in the University of Toronto Medical Alumni was reported at the annual meeting. Scholarships and bursaries to the amount of $800 are being paid yearly. A new graduate bursary fund in Psychiatry has been announced.

The Association entertained the class of 1898 and the class of 1948 at a banquet at the Royal York in June. Twelve members of the class of '98 were present.

A postgraduate course is being put on by the Association at Sunnybrook Hospital on November 11, 12 and 13. The Varsity-Western Rugby game is to be on the afternoon of the 13th.

LILLIAN A. CHASE

Quebec

Au début de septembre, les médecins de langue française de l'Amérique du Nord se sont rendus en grand nombre à Ottawa et à Hull, où se tenait le dix-neuvième congrès de leur Association. Outre les réunions scientifiques, tenues au Château-Laurier, ce congrès était rehaussé d'une exposition d'art, de nombreuses projections cinématographiques, d'une réception à l'Ambassade de France, et se clôturait par un dîner et un bal au Château-Laurier.

Les professeurs Donald-A. Hingston et Urgel Gariépy, quittant les chaires de clinique chirurgicale à l'Université de Montréal, sont élus professeurs émérites. Le professeur J.-A. Jarry, qui occupait la chaire de phtisiologie, a été élu à la même dignité.

Le docteur Paul Bourgeois, chef du service d'urologie à l'hôpital Notre-Dame, représentera la Canadian Urological Association, dont il est le trésorier, à l'Association internationale d'urologie.

Le docteur Mercier Fauteux, de Montréal, a assisté aux Journées médicales de Bruxelles, où il représentait les universités montréalaises. Au cours de son voyage, il a visité les centres de chirurgie cardiaque de France et de Suède.

PAUL DE BELLEFEUILLE

Saskatchewan

Dr. G. Gordon has left Rosetown to reside in Chilliwack, B.C., due to ill health. His practice has been taken over by Dr. G. G. K. Graham and Dr. C. R. Giles.

The Moose Jaw and District Medical Society, through its staff meetings at the Moose Jaw General Hospital has been considering ways and means of increasing the hospital accommodation in the City of Moose Jaw. The shortage has been acute resulting in long delay for some patients. It was noted that the last addition to the Moose Jaw General Hospital was made in 1948.

After serious consideration and in order to get started, a citizens' meeting was called under the auspices of the Moose Jaw Medical Society. At this meeting a committee was established to open a campaign to collect funds to assure the community that adequate hospital facilities would be built.

The initiative of the physicians of Moose Jaw in calling this citizens' meeting and starting action on their new hospital, is to be commended.

The Council of the College met recently in North Battleford guests of the Sisters of Notre Dame Hospital and the member for that District, Dr. J. J. Hamelin. The doctors were very impressed with the very modern and beautifully appointed addition to the hospital. It has been constructed with the view of being a real health centre for the district.

The Council of the College at its recent meeting, taking note of the retirement of Dr. R. G. Ferguson, and the very fine contribution he has made to the profession, conferred upon him an Honorary Membership in the College. Dr. Ferguson, through his leadership in the attack on tuberculosis has succeeded in establishing the Province of Saskatchewan as a model to other countries.

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General

American Board of Ophthalmology. Candidates for the certificate of the American Board of Ophthalmology are accepted for examination on the evidence of a written qualifying test. These tests are held annually in various parts of the United States. Registration is already closed for the next test to be given in January, 1949. Applications are now being accepted for the 1950 written test. They will be considered in order of receipt until the quota is filled.

Practical examinations for acceptable candidates, 1949: San Francisco, March 21 to 24; New York, June 11 to 15; St. Louis, October 15 to 19; Boston, December. Apply Dr. S. Judd Beach, Executive Office, Cape Cottage, Maine.

The Twelfth British Congress of Obstetrics and Gynæcology. This meeting will be held in London, England, July 6 to 8, 1949.

Owing to the difficulties that exist at the present time in arranging hotel accommodation, travel, etc., the Hon. Secretaries would like to have the names of those who hope to attend by March 31, 1949 at the latest, and, if possible, very much before that date. Ian Jackson, Hon. Sec'y., 58 Queen Anne St., London, W. 1.

The American Goitre Association will meet in the Hotel Loraine, Madison, Wisconsin, May 26, 27, and 28, 1949. The program for the three day meeting will consist of papers dealing with goitre and other diseases of the thyroid gland, dry clinics and demonstrations.

National Research Council Grants 31 Medical Fellowships. Medical Fellowships have been awarded by the National Research Council to 31 graduates in medicine to enable them to pursue postgraduate research during 1948-49. The grantees represent ten different universities, including one in Australia and one in England. The fellowship holders will carry on their research investigations at eight universities. Six of these research centres are located in Canada, one in England, and one in Zurich, Switzerland. Distribution of the awards by the universities at which they will be held is as follows: McGill, 8; Montreal, 1; Queen's, 4; Toronto, 7; Western Ontario, 8; Manitoba, 1; Oxford, England, 1; Zurich, Switzerland, 1.

Medical Fellowships vary in amount from $1,500 to $2,500, depending on the previous training and experience of the candidates. The Division of Medical Research of the National Research Council also makes Grants-in-Aid to Canadian universities for medical research studies on approved subjects. During the present year 118 such grants valued at $264,173, are being held. Nominal value of the 31 medical fellowships awarded this year is $64,600.

In the following table are shown the geographical distribution of the Medical Research Fellowship holders, grouped according to their home towns, and showing the universities from which they graduated, the universities at which they will carry on their research, and the branch of science in which they will pursue their studies:

GEOGRAPHICAL DISTRIBUTION OF MEDICAL RESEARCH FELLOWSHIP WINNERS

Grouped according to their home towns and showing university of graduation, place at which each will do his research, and his subject of study.

Home town	*Name of Fellow*	*Graduated from*	*To work at*	*In*
Quebec				
Lachine	McMillan, Dr. G. C.	McGill	Zurich	Pathology
Montreal	Kobernick, Dr. S. D.	McGill	McGill	Pathology
	Nunes, Dr. Doris S.	McGill	McGill	Bacteriology
	Payne, Dr. T. P. B.	McGill	McGill	Pathology
	Phaneuf, Dr. Jean	Ste. Marie College	Montreal	Medicine and Surgery
	Waugh, Dr. D.	McGill	McGill	Pathology
Westmount	Finkelstein, Dr. W. E.	McGill	McGill	Pathology
	Mackenzie, Dr. K. R.	McGill	Royal Victoria Hospital	Medicine
Ontario				
Belleville	Younghusband, Dr. O. Z.	Queen's	Queen's	Pathology
Brownsville	Cook, Dr. W. H.	Western Ont.	Western Ont.	Anatomy
Chatham	Stewart, Dr. H. B.	Toronto	Toronto	Biochemistry
London	Buck, Dr. R. C.	West rn Ont.	Western Ont.	Biochemistry
	Hatcher, Dr. J. D.	Western Ont.	Western Ont.	Physiology
	Heagy, Dr. F. C.	Western Ont.	Western Ont.	Biochemistry and Bacteriology
	Loynes, Dr. J. S.	Queen's	Western Ont.	Pharmacology
	McNabb, Dr. A. R.	Western Ont.	Western Ont.	Biochemistry
	Teasdall, Dr. R. B.	Western Ont.	Western Ont.	Physiology
Parry Sound	Little, Dr. J. A.	Toronto	Toronto	Biochemistry
Pembroke	Bresnaham, Dr. T. J.	Queen's	Queen's	Medicine
St. Catharines	Taylor, Dr. N. B. G.	Toronto	Western Ont.	Medicine
Toronto	Fraser, Dr. D.	Toronto	Toronto	Medicine
	Fitzgerald, Dr. J. P.	Cambridge	Queen's	Pathology
	Hutcheon, Dr. D. E.	Toronto	Oxford	Pharmacology
	Kalant, Dr. N.	Toronto	Toronto	Medicine
	Slade, Dr. H. C.	Dalhousie	Toronto	Medicine
	Watt, Dr. J. G.	Toronto	Toronto	Medicine
Tweed	Elliott, Dr. S. M.	Queen's	Queen's	Medicine
Saskatchewan				
Aberdeen	Wilkinson, Dr. G. W.	Saskatchewan	McGill	Experimental Surgery
Assiniboia	Gemmel, Dr. J. R.	Manitoba	Manitoba	Medicine
British Columbia				
Victoria	Johnston, Dr. M. W.	Saskatchewan	Toronto	Medicine
Australia				
Sydney	Hunter, Dr. J. M.	Sydney	McGill	Neurology

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A report entitled Medical and Hospital Services Provided under Prepayment Arrangements, Trinity Hospital, Little Rock, Ark., 1941-42 has been prepared by Margaret C. Klem, Helen Hollingsworth and Zelma A. Miser. It covers a study of physicians' care in the office, home and hospital and hospitalization received by subscribers and their dependents under prepayment arrangements, and extends over a period of two years.

The report is for sale at the Government Printing Office, Washington, D.C., price $1.00. A preliminary report by Margaret Klem for the first year on the same subject is also available at 5c a copy.

National Research Council Appoints Subcommittee on Oncology. The Committee on Pathology of the National Research Council has appointed a Subcommittee on Oncology. The members are: Dr. Shields Warren, Chairman; Dr. Balduin Lucké; Dr. Fred Stewart; Dr. Harold Stewart; Dr. Arthur P. Stout; Dr. Milton C. Winternitz; Dr. Howard T. Karsner, Chairman of Committee on Pathology, *ex-officio*.

The objectives of the Subcommittee are: (1) Improvement in the teaching of oncology; (2) Dissemination of information on oncology to clinical pathologists, students and teachers of oncology; (3) The establishment of criteria for diagnosis of tumours; (4) The simplification of terminology by recommending a single term for each tumour and listing separately the appropriate synonyms.

The Subcommittee expects to work with existing agencies to promote clarity and unity in tumour nomenclature and classification.

Urology Award. The American Urological Association offers an annual award of $1,000.00 (fiirst prize of $500.00, second prize $300.00 and third prize $200.00) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to residents in urology in recognized hospitals. For full particulars write the Secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis 3, Tennessee. Essays must be in his hands before February 15, 1949.

BOOK REVIEWS

Rorschach Study of the Psychological Characteristics of Alcoholics. C. Buhler, Consulting Psychologist, Los Angeles County General Hospital, and D. W. Lefever, Professor of Education, University of Southern California. 64 pp. $0.75. Hillhouse Press, New Haven, Connecticut, 1948.

This small monograph is the sixth of a series of studies on alcohol from the Yale Laboratory of Applied Physiology and has been reprinted from the *Quarterly Journal of Studies on Alcohol.* It is also part of a larger study intended to establish more effective criteria for Rorschach interpretation. To the average medical reader the more technical portion of this report, dealing with Rorschach signs and their graphic analysis, will hold little interest and will prove difficult to follow. The study is controlled with non-alcoholic groups, and the alcoholics are subdivided into several diagnostic categories. The conclusions of the authors are interesting and deserve further study. In the alcoholic individual, it is felt, anxiety results from loss of control in acute tension-producing situations. He is unable to stand this acute anxiety and immediately escapes into alcohol. This alcoholic refuge from acute tension is not comparable to the symptoms of a psychoneurosis; these latter are reactions to deep-level conflicts of long standing. Both mechanisms may be at work in the same individual, however. Further, the alcoholic differs from the psychopath in that he escapes with a bad conscience. This small booklet presents well the thesis of the authors. It should be of interest to the average medical reader as well as to the specialist.

Bilharzial Cancer. M. A. Afifi, Former Director of the Radiological and Electrotherapeutic Departments of the Egyptian Government Hospital, Alexandria. 111 pp., illust. 16s. H. K. Lewis & Co. Ltd., London, 1948.

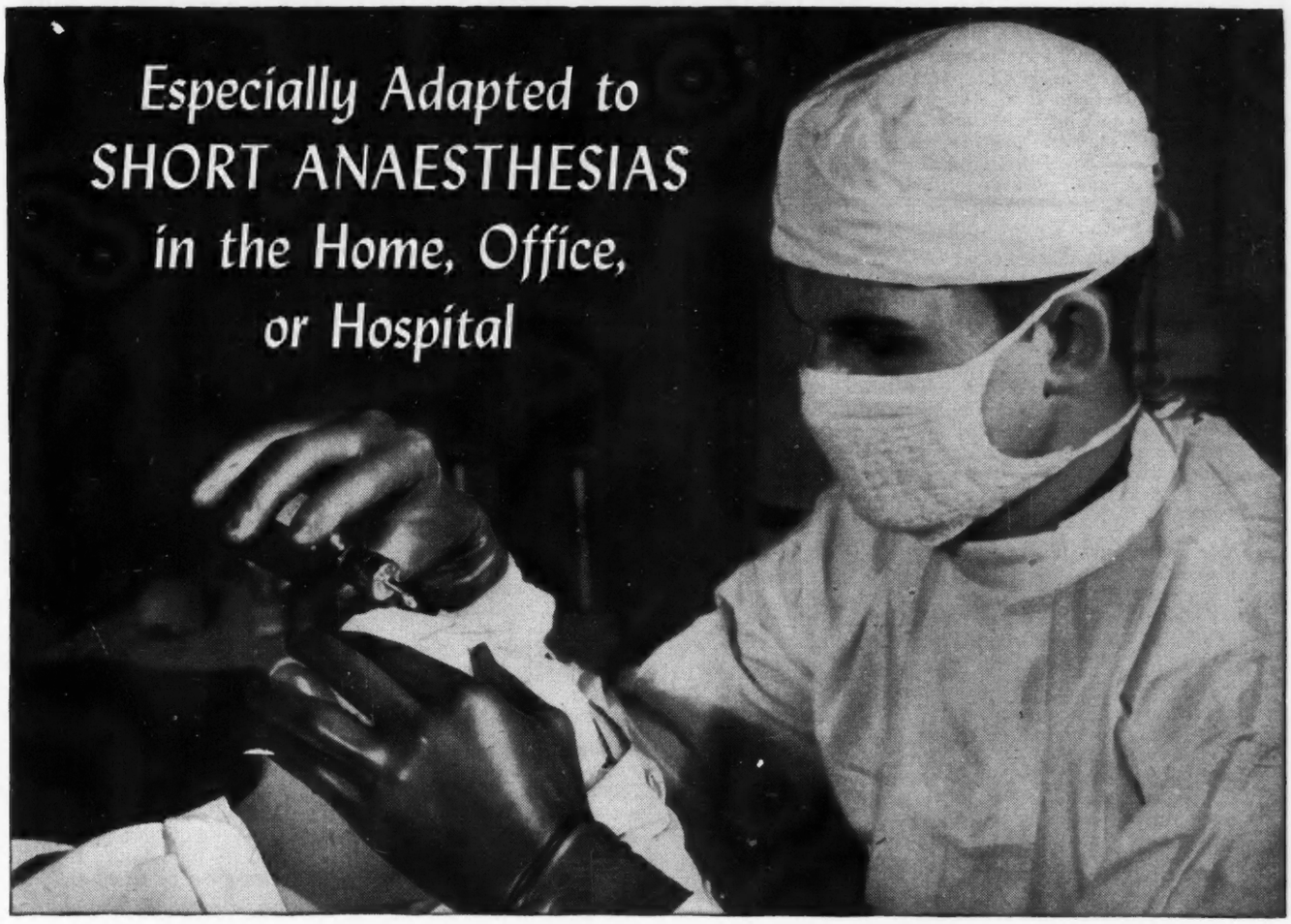
This little book makes interesting reading. Dr. Afifi's long experience with the diagnosis of cancer associated with bilharzial infection strongly suggests a cause and effect relationship between the two diseases. The bladder and lower bowel are the sites in which the association is generally found. When the two diseases coexist treatment becomes a problem. Surgical interference involves the risk of sepsis greater than usual because of the condition of the tissues due to bilharzial infection. Radiological methods therefore offer the best hope of cure provided they can be applied. A foreword by Sir Alfred Webb-Johnson strongly recommends the book. This recommendation seems justified and the work should be of help to radiologists and surgeons working in regions where bilharziasis is a common disease.

Anatomy of the Bronchial Tree. R. C. Brock, Surgeon to Guy's Hospital. 98 pp., illust. $12.00. Oxford University Press, London, New York, Toronto; McAinsh & Co. Ltd., Toronto, 1947.

This monograph, a reprint of previous publications by the same author, with some additions, deals with the anatomy of the bronchi and the pulmonary segments. The practical application of this knowledge to the radiological localization and surgical approach of lung abscesses is also discussed. It is based on the dissections of normal and diseased lungs, metallic casts of the bronchi, bronchograms and post-mortem specimens. The book is written simply and lucidly and is profusely illustrated with photographic reproductions of the authors' dissections and casts, diagrams and bronchograms, as well as ordinary radiographs of the lungs. These make it easy to follow the text. There is also a short chapter on the level of the lung fissures and one on supernumerary and anomalous bronchi. This is the first book to appear on bronchopulmonary segmentation and its clinical application in the investigation of localized pulmonary disease. As such it should be of interest to every thoracic surgeon, physician, radiologist and bronchoscopist.

Practical Clinical Psychiatry. E. A. Strecker, Professor of Psychiatry, School of Medicine, University of Pennsylvania; F. G. Ebaugh, Professor of Psychiatry, University of Colorado, School of Medicine; and J. R. Ewalt, Professor of Neuro-Psychiatry; Director, Galveston State Psychopathic Hospital, University of Texas Medical Branch. 476 pp., illust., 6th ed. $5.00. The Blakiston Co., Philadelphia and Toronto; Doubleday Publishers, Toronto, 1947.

This sixth edition of what has become the recognized American textbook for students of psychiatry in United States, has retained all its former virtues of clarity and easy reading with adequate scope to cover the subject. The book has been brought up to date with the general and steady advances made in psychiatry since the fifth edition in 1940. In the introduction and where indicated, references are made to the psychiatric problems highlighted by either the acute or extensive problems of the past war. In future editions it is hoped that the authors will have more to offer in the prevention and treatment of those mental disorders associated with old age. The book is recommended as a satisfactory standard text that fills a definite need and the authors are to be congratulated on maintaining its character throughout the years.



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Clinical Diagnosis by Laboratory Methods. J. C. Todd, Late Professor of Clinical Pathology, University of Colorado School of Medicine; A. H. Sanford, Professor of Clinical Pathology, Mayo Foundation, University of Minnesota, with the Collaboration of G. G. Stilwell, Division of Clinical Laboratories, the Mayo Clinic. 954 pp., illust., 11th ed. $8.25. W. B. Saunders Co., Philadelphia and London; McAinsh & Co., Ltd., Toronto, 1948.

This standard manual of clinical pathology has been considerably revised and extended. In particular, a full chapter is now devoted to medical mycology, while considerable additional material has been included in the chapters on serodiagnostic methods and on hæmatology. This manual provides reasonably complete coverage of the practical aspects of routine clinical laboratory procedures. It should be borne in mind, however, if this book is being used as a guide for the installation of a laboratory procedure, that in some cases, newer, simpler techniques are available, which offer advantages over the older routine methods.

Common Contagious Diseases. P. M. Stimson, Associate Professor of Clinical Pædiatrics, Cornell University Medical College. 503 pp., illust., 4th ed. $4.00. Lea & Febiger, Philadelphia; Macmillan Co. of Canada, Toronto, 1947.

This book will be a very useful addition to the general practitioner's library and should be a helpful volume for any student. It is well-written and easy to read. The first three chapters take up the principles of contagion, serum reactions and the use of sulfonamides in the common contagious diseases. The main contagious diseases, common to this country, have been fully discussed. The treatment and prophylactic measures have been well outlined.

Fundamentals of Human Reproduction. E. L. Potter, Associate Professor of Pathology, Department of Obstetrics and Gynæcology, School of Medicine, University of Chicago. 231 pp., illust. $3.50. McGraw-Hill Book Co., Inc., New York, Toronto and London, 1948.

In her treatment of this subject, Dr. Potter has broken away from the usual textbook style and she presents the miracle of birth in a dramatic fashion which is also very readable. The first section of the volume includes a study of early forms of animal life, the general pattern of reproduction and the factors responsible for body form. Succeeding chapters deal with all phases of human reproduction, from the anatomy of the sex organs through a discussion of the processes concerned with the development of each organ of the human body. Scientific terminology has been simplified to a remarkable degree thus enhancing the value of the book as background reading. Dr. Potter has taught nurses for many years and her book has been written with a view to assisting student nurses in the study of obstetrics. It will, however, be welcomed by interns as well as by those general readers who are interested in human origins.

Hospital Care of Neurosurgical Patients. W. B. Hamby, Professor of Neurology and Neurological Surgery, University of Buffalo School of Medicine, Buffalo. 156 pp., 2nd ed. $4.00. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto.

Specific directions for the management of neurosurgical patients are given most of the consideration in this manual. Interns and nurses, however, will find much in the book which may be of help in caring for all types of patients. The section headed, "General Considerations", might well form the basis for the instructions to be given housemen at the start of their internship. The author briefly reviews the anatomy which pertains to the central nervous system; follows this by concise instructions in diagnostic procedures; then in tabular and summary form outlines methods for the care of specific neurosurgical disorders. Emphasis is placed on the possible development of complications and the recognition of complications once they have occurred. It is stated that housemen should themselves be able to estimate the quantities of albumen and globulin in samples of cerebrospinal fluid, but one wonders if these complicated laboratory procedures can be carried out by untrained interns. Contrary to the present day teachings in many centres, large amounts of heat are still recommended for shocked patients. Hospital libraries would do well to add this book to their shelves for the guidance of interns.

Human Physiology. F. R. Winton, Professor of Pharmacology, University College, London; and L. E. Bayliss, Reader in Physiology, University College, London. 592 pp., illust., 3rd ed. $8.00. The Blakiston Co., Philadelphia and Toronto, 1948.

The third edition of this text is an improvement over previous versions. Because of the format it is actually shorter than would appear at first sight. Designed for first year medical students it is an adequate short text but will not satisfy the inquiring mind.

Identification of Tumours. N. C. Foot, Professor of Surgical Pathology, Cornell University Medical College. 397 pp., 241 illust. $7.50. J. B. Lippincott Co., New York, Montreal, 1948.

This should prove to be a very useful volume for the busy surgical pathologist. The material is very concisely and conveniently arranged under the various systems and organs. There are numerous illustrations which are of good quality and are well placed in reference to the text. The whole field of pathology of tumours is covered in 390 pages, including a chapter on technical methods of fixing and staining, and a ready reference table for the tentative identification of neoplasms.

Operative Gynæcology. H. S. Crossen, Professor Emeritus of Clinical Gynæcology, Washington University School of Medicine; and R. J. Crossen, Assistant Professor of Clinical Gynæcology and Obstetrics, Washington University School of Medicine. 999 pp., illust., 6th ed. $16.50. C. V. Mosby Co., St. Louis, Mo.; McAinsh & Co. Ltd., Toronto, 1948.

The sixth edition of this well known work which was first published in 1915 appears ten years after the fifth edition. As in that time there have been advances and discoveries, this edition is entirely revised and reset. The aim of the senior author from the first has been to be a guide to the operator, not merely in describing the successive steps of an operation, but in selecting the particular operation best suited to the patient. In this edition the authors, father and son, have two further aims, the extension of practical means for the prevention of cancer of the ovaries, uterus and external genitals, and the development of measures for giving more effective local relief to patients with general handicaps which contraindicate operative removal of the seriously troublesome pelvic lesion. To this end mention is made of the application of radium and deep x-ray therapy and of the administration of sex hormones. The medico-legal aspects of operative gynæcology and the insistence on the necessity of the utmost care to prevent a foreign body being left in the abdominal cavity are worthy of note.

A few omissions are noted; *e.g.*, the Pomeroy method of sterilization is not mentioned, disgerminoma is not included among the ovarian tumours, there is no reference to the peritoneoscope, the culdescope or pneumoperitoneum in diagnosis of obscure pelvic conditions. All in all the book can be trusted to give carefully reasoned, clearly phrased selective guidance.